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UCRL-53953

Baseline Public Health Assessment for CERCLA Investigations at the LLNL Livermore Site

Technical Editors

D. W. Layton
J. I. Daniels
W. F. Isherwood

Contributing Authors

K. T. Bogen
R. T. Cederwall
J. I. Daniels
M. D. Dresen*
K. Goyal
C. H. Hall
L. C. Hall
V. M. Johnson
D. W. Layton
B. J. Mallon
P. F. McKereghan*
T. E. McKone
E. M. Nichols*
D. W. Rice, Jr.
M. C. Small*
R. K. Thorpe
A. F. B. Tompson
F. A. Yukic*

September 1990

***Weiss Associates, Inc., Emeryville, California**

Environmental Restoration Division

LAWRENCE LIVERMORE NATIONAL LABORATORY
University of California • Livermore, California • 94551

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Preface

Lawrence Livermore National Laboratory (LLNL) is a U.S. Department of Energy facility operated by the University of California. In 1987, the LLNL Livermore site was officially placed on the National Priorities List as a Federal Superfund site. As a means of coordinating the requirements of the different State and Federal agencies with oversight functions and ensuring that the site remediation efforts proceeded in a timely and effective manner, a Federal Facility Agreement was completed that defined the responsibilities of the relevant parties. As part of that agreement, a Baseline Public Health Assessment (BPHA) was identified as a required document.

The BPHA was originally prepared and submitted as a draft document in June 1989 to the State and Federal agencies with oversight functions and also was made available for public comment. The material contained in that draft document then underwent substantial revision in response to the comments that were received. This revised BPHA material was then incorporated into Sections 5 and 6, primarily, and the corresponding appendices of the *CERCLA Remedial Investigations Report for the LLNL Livermore Site* (RI)*.

This stand-alone version of the BPHA was reconstructed from the revised BPHA material presented in the final version of the RI report. The RI document (with this BPHA material) was formally accepted in May 1990 by the State and Federal agencies with oversight responsibilities.

* Thorpe, R. K., W. F. Isherwood, M. D. Dresen, and C. P. Webster-Scholten, Eds. (1990), *CERCLA Remedial Investigations Report for the LLNL Livermore Site*, Lawrence Livermore National Laboratory, Livermore, Calif. (UCAR-10299).

Executive Summary

Introduction

In 1987, the U.S. Environmental Protection Agency (EPA) added the Lawrence Livermore National Laboratory (LLNL) Livermore site to the National Priorities List (NPL) due to volatile organic compounds (VOCs) found by LLNL in ground water onsite and offsite. One key component of the site cleanup effort at an NPL or Superfund site is the Remedial Investigation (RI), which includes studies and monitoring programs to acquire and analyze pertinent site-related data, such as the nature and extent of contamination and the characteristics of the local hydrogeology. An important part of the RI is the Baseline Public Health Assessment (BPHA), which addresses the potential future public health risks that could exist if no cleanup is attempted. This BPHA material was included in the RI for the LLNL Livermore site*, which was submitted to regulatory agencies in May 1990. The BPHA is published here as a stand-alone document for the convenience of those interested only in this material.

Because the U.S. Department of Energy (DOE), LLNL, and environmental regulatory agencies are dedicated to the remediation of contaminated soils, sediments, and ground water at the Livermore site, the potential risks described herein are unlikely to occur. This BPHA provides the information needed to evaluate the benefits of cleanup alternatives.

The primary parts of this BPHA are:

- A screening analysis to determine which contaminants and potential exposure pathways should be the focus of the BPHA.
- Simulations of the transport and fate of the ground water contaminants.
- An assessment of potential human exposures to contaminants in ground water and soil.
- Estimation of the health risks associated with the predicted exposures.

In the following sections, we summarize the principal findings of each of those analyses and assessments.

Contaminant Screening for Hazard Assessment

A screening analysis was conducted to determine which substances and exposure pathways are potentially important from the perspective of adverse health effects for the public offsite and employees onsite. To accomplish this goal, we statistically analyzed chemical data for thousands of water samples from monitor wells and data from several different soil/sediment and storm-water runoff sampling studies.

The first part of the screening focused on the VOCs in ground water. These are dominated by trichloroethylene (TCE), tetrachloroethylene (PCE), and chloroform, which account for an

* Thorpe, R. K., W. F. Isherwood, M. D. Dresen, and C. P. Webster-Scholten, Eds. (1990), *CERCLA Remedial Investigations Report for the LLNL Livermore Site*, Lawrence Livermore National Laboratory, Livermore, Calif. (UCAR-10299).

estimated 91% of the total amount of VOCs dissolved in ground water. Based on the results of animal bioassays involving chronic exposure, lifetime exposure of humans to TCE, PCE, and chloroform may pose a cancer risk. The remaining 9% of the VOCs in ground water is composed of a variety of organic compounds, over half of which is 1,1-DCE. From a health-risk perspective, however, the most hazardous is considered to be carbon tetrachloride, which we use to represent the adverse effects of the remaining 9% of the VOCs.

The second phase of the screening examined other contaminants in ground water, including benzene from the gasoline spill, radioactive tritium, and inorganic substances. The very slow ground water movement in the locale of the gasoline spill, combined with benzene's fairly rapid biodegradation rate, virtually assures that benzene will never reach detectable concentrations beyond DOE property. Consequently, benzene does not constitute a public-health threat. Tritium exceeds the State of California and Federal drinking water standard of 20,000 pCi/L in one well (approximately 25,000 pCi/L). Levels have declined significantly over the period of our sampling due to radioactive decay and will be reduced to well below the standard prior to offsite migration in ground waters. Continued monitoring will be used to verify this conclusion.

Several inorganic substances, including nitrate, sulfate, manganese, and chromium, exceed applicable regulatory limits in various monitor wells. Elevated concentrations of the inorganic compounds were found sporadically in wells onsite and offsite. However, we found no evidence to associate these compounds (with the possible exception of chromium) with potential sources at the Livermore site. In fact, there is substantial evidence that the observed concentrations are indicative of the normal variation in the background levels of such constituents in ground waters in the Livermore Valley.

The final portion of our screening analysis examined the nature and extent of soil contamination onsite and offsite. Principal findings of that analysis are that:

- The public is not directly exposed to contaminated soils because soil samples taken in offsite arroyos draining from the Livermore site are free of contamination.
- The only potential soil-based exposure pathway for the public is through the inhalation of VOCs volatilized from soil onsite, which is shown to be insignificant.
- The number of persons onsite exposed to VOCs and other organic compounds, as well as inorganic chemicals detected in soils, is insignificant, based on our evaluation using the EPA recommended procedure for assessing noncarcinogenic health hazards and health risks related to carcinogenic effects.
- No evidence in the data reviewed for the BPHA suggests that soil concentrations of radioactive substances present a health hazard to adults onsite or to the public offsite.

Contaminant Migration

To assess the potential future health risks of the known contaminants in ground water, we modeled the movement of VOCs from their current distributions. We calculated 70-y average concentrations at both existing and potential wells offsite using an accepted analytical model of contaminant transport and fate in the ground water system. The model accounts for the four physical and chemical processes that affect the movement and distribution of contaminants.

These are:

1. Advection (transport by bulk movement of water).
2. Dispersion (transport by mechanical spreading and molecular diffusion).
3. Retardation (sorption by solid materials).
4. Degradation (biological or abiotic chemical transformation of a contaminant).

Sensitivity analyses showed degradation to provide the dominant uncertainty in predicted future concentrations, assuming the 70-y exposure interval could be taken about any point in time. By this criterion, the ground water velocity was of less importance than the total amount of contaminant introduced to the ground water. Accordingly, our source terms accounted for contaminants already dissolved in the ground water, as defined by our subsurface sampling program, and also for sorbed contaminants that might be added to the water on a delayed basis. The latter represented material held in the saturated sediments below the water table, plus material that may not yet have reached the water table from the vadose zone. We used conservative values for the sorbed contaminant masses, based on preliminary results of our ongoing source investigation sampling program.

The physical parameters describing these four processes depend on the hydrogeology and environmental chemistry, and, therefore, may vary over the complete ground water flow path. The path, in turn, is determined by the direction of ground water flow and the locations of potential receptor points—either existing water supply wells or hypothetical monitor wells. Despite comprehensive characterization of the ground water, there remains some uncertainty as to the total mass of contaminants trapped in unsaturated sediments that could reach ground water.

We treat these uncertainties by defining two scenarios: (1) a "best-estimate" case, consisting of private and municipal supply wells as receptors and the most probable hydrogeologic, chemical, and source parameters; and (2) a "health-conservative" case that considers hypothetical receptors very near the LLNL site (i.e., potential monitor wells), the highest possible source contamination, and hydrogeologic parameters that would produce the highest offsite concentrations. For the health-conservative case, we assume there is no natural degradation of the compounds, no dilution with clean water at a receptor, and the fastest plausible transport rate. In both cases, we assume all the ground water moves westward, directly toward the receptors, when in fact, much of the ground water flows towards more distant potential receptors to the northwest.

Table Ex. Sum.-1 lists the maximum lifetime concentrations of total VOCs at a receptor for the two scenarios. To demonstrate the extremely slow transport velocity, we also include the time at which the highest concentration would occur. In the best-estimate case, the highest 70-y average concentration would be 0.15 ppb in 270 y, which is essentially below current detection limits. However, the health-conservative scenario yields maximum concentrations well above present action levels for several individual compounds.

Table Ex. Sum-1. Summary of maximum lifetime 70-y average concentrations of total VOCs at receptor wells and corresponding increased incremental cancer risk.

Scenario	Highest 70-y average concentration of total VOCs (ppb)	Arrival time of maximum concentration (y)	Cancer risk
Best estimate[a]	0.15	270	2×10^{-7}
Health conservative estimate[a]	440	110	1×10^{-3}
Health conservative estimate[b]	584	35	2×10^{-3}

[a]Based on receptor wells in downtown Livermore.

[b]Based on a potential monitor well drilled 250 ft west of LLNL.

Exposure and Health Risks

Exposures to predicted concentrations of VOCs in well water are governed principally by the potential uses of existing or future wells. Our review of the wells located west of the Livermore site indicates that domestic and irrigation uses are the most significant. Accordingly, we estimate potential exposures to VOCs for tap water uses in the home via ingestion of water, inhalation of VOCs volatilized during showers, and dermal uptake of VOCs in bath water. Potential irrigation-related exposures consist of the ingestion of fruits and vegetables from home gardens watered with contaminated well water and the inhalation of VOCs that volatilize during sprinkler irrigation.

The exposure assessment demonstrates that tap water use is the main exposure pathway and that water ingestion accounts for a significant proportion of the daily predicted dose rate. Inhalation of household air containing VOCs derived from tap water represents another significant fraction of the daily dose rate, and dermal uptake also accounts for a significant amount of the total intake. The exposure from ingestion of fruits and vegetables irrigated with well water contaminated with VOCs is shown to be less than the exposures estimated for tap-water uses in a home. Inhalation exposures to VOCs volatilized from sprinklers are found to be several orders of magnitude below those estimated for the other water-based pathways.

The incremental risk of developing cancer as a result of the predicted 70-y (lifetime) exposure to a VOC in well water is calculated as the product of the estimated dose rate and a carcinogenic potency. Potencies for the VOCs of interest were obtained by fitting a dose-response model to tumor-incidence data from animal bioassays involving chronic exposure. The maximum cancer risk associated with the best-estimate case for the combined 70-y maximum exposure to VOCs at a municipal supply well in downtown Livermore is calculated to be 2×10^{-7} (an additional 2 in 10 million chance of developing cancer over a lifetime of exposure). Under the health-conservative transport scenario, the highest predicted risk is 2×10^{-3} for exposure to contaminants in water from a potential monitor well drilled 250 ft west of the Livermore site. Because no wells are likely to be drilled within this area, we also show the risk based on the downtown Livermore receptor sites under the health-conservative transport scenario. This scenario results in a maximum risk of 1×10^{-3}.

Conclusion

In summary, we emphasize that all of these analyses of risk are based on the premise that no remediation will occur. In fact, LLNL will remediate these contaminants and actual risks will be much lower. Moreover, the risks that are reported are estimates based on a number of assumptions, and for this reason the risk may be as high as predicted or may be considerably lower. We also stress that *no* members of the public are currently exposed to VOCs derived from the use of wells near the LLNL site.

1. Introduction

D. W. Layton

During the period 1980 to 1983, monitor wells were drilled at the Lawrence Livermore National Laboratory (LLNL) Livermore site as part of an effort to determine whether local ground water was contaminated. This effort was motivated partly by evidence suggesting that previous operations at the Livermore site had resulted in releases of hazardous materials. Chemical analyses of water samples from the nearby private wells indicated that four wells supplying potable water to residences not connected to municipal water supplies were contaminated with volatile organic compounds (VOCs). The VOCs of concern included solvents such as trichloroethylene (TCE) and tetrachloroethylene (PCE). These compounds are considered hazardous to human health primarily because they have been shown to cause cancer in laboratory animals. To protect the health of the affected residents, LLNL immediately supplied bottled water to them, and later connected the occupied houses to municipal water supplies. These preliminary investigations and corrective actions represent the initial activities in what has become a major effort to investigate the nature and extent of ground water and soil contamination at the Livermore site, to assess the potential health risks of that contamination, and to evaluate alternative cleanup options and technologies.

1.1. Background

State and Federal agencies overseeing work at the Livermore site have included the California Department of Health Services (DHS); the California Regional Water Quality Control Board (RWQCB), San Francisco Bay Region; and the U.S. Environmental Protection Agency (EPA), Region IX. The DHS was involved at an early stage in the investigations because of concern over public exposures to hazardous substances in well waters and potential health effects. It issued an Order of Compliance in 1984 that dealt with the measures to be taken to prevent exposures to contaminated well water as well as with the development of a site investigation plan. The RWQCB later issued Order 85-134 in 1985 to guide the investigative efforts involving ground water contamination. Subsequent Orders have been issued by the RWQCB as the site investigations have progressed. In 1987, the EPA placed the Livermore site on the National Priorities List (NPL). The remediation of NPL sites is governed by provisions of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), as amended by the Superfund Amendments and Reauthorization Act (SARA) of 1986. Implementation of CERCLA along with applicable State laws at the Livermore site is carried out via a Federal Facility Agreement that specifies the obligations of the U.S. Department of Energy (DOE), LLNL, and the different State and Federal agencies with regulatory responsibilities involving site remediation.

Principal parts of CERCLA-mandated cleanup programs are a Remedial Investigation (RI) and a Feasibility Study (FS). The RI consists of various studies designed to acquire pertinent data on the characteristics of the site (e.g., sources of contaminant releases, extent of contamination, exposure pathways, and hydrogeology). The FS is devoted to the analysis of the

health, environmental, technical, and economic aspects of alternative cleanup options and relies heavily on the results of the RI as the principal source of background data on the site. Together, the RI/FS efforts provide the information necessary to arrive at decisions regarding which remedial actions offer the most promise for achieving a successful cleanup of residual contamination at the site.

Note that the work for this BPHA was completed in 1990 and, consequently, the data upon which it is based were frozen in early 1990. Subsequent work and data collected may slightly modify some of the analyses herein.

1.2. Baseline Public Health Assessment

This document addresses the potential for future public-health risks that could exist if no cleanup were attempted at the LLNL site. Under this scenario, we assume that no attempt is made to either mitigate or prevent exposures to toxic substances. In essence, the assessment serves as a baseline case that can be used to compare the relative effectiveness of alternative remediation strategies in reducing public-health risks. It is important to keep in mind, however, that the DOE, LLNL, and the environmental regulatory agencies are dedicated to the remediation of contaminated soils, sediments, and ground water at the LLNL site. Given this perspective, this study provides the information that is required to evaluate the benefits of cleanup alternatives. (This report uses the term "soil" only for the uppermost meter or so, where surface oxidation has taken place. Below this, we refer to the unconsolidated gravels, sands, silts, and clays as "sediment.")

Pertinent information on the content and preparation of health assessments at Superfund sites is contained in *Guidance for Conducting Remedial Investigations and Feasibility Studies Under CERCLA* (U.S. EPA, 1988a), the *Superfund Exposure Assessment Manual* (U.S. EPA, 1988b), and the *Superfund Public Health Evaluation Manual** (U.S. EPA, 1986). The primary components of the current study are outlined in Figure 1-1.

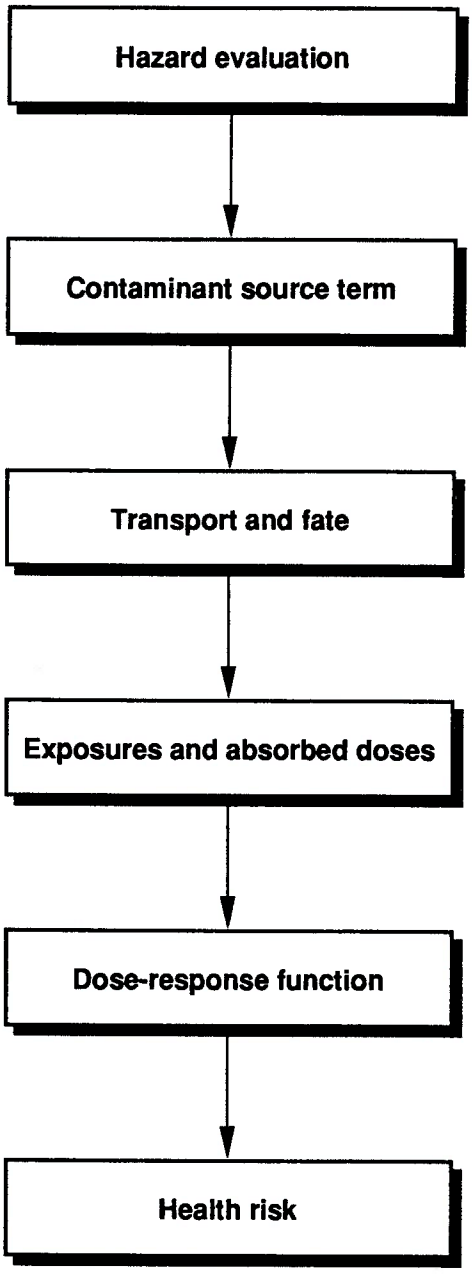
The first task of the baseline risk assessment is to evalutate hazards posed by the contaminants detected in the environmental media (e.g., soils, ground water, and air) at the site. This evaluation identifies those contaminants that *potentially* pose the greatest risks to human health. Once the relevant set of contaminants is defined, we simulate their migration in ground water to selected receptor wells and use those results to assess exposure. Finally, we assess the public health risk resulting from that exposure.

1.3. Organization of the Baseline Public Health Assessment

Section 2 of this BPHA provides an overview of past and present investigations involving the characterization of soil and ground water contamination at the Livermore site. In Section 3, we

* The most recent guidance document from the EPA for preparing a baseline public health assessment (U.S. EPA, 1989), which replaces the *Superfund Public Health Evaluation Manual* (U.S. EPA, 1986), was not published until well after the draft version of this BPHA was submitted for review.

present the screening analysis we performed to determine which compounds should be the subject of the health-risk assessment. Subsequent sections deal with the transport of VOCs in ground water (Section 4), quantification of human exposures to contaminants, and prediction of health risks (Section 5). Appendices supporting the various sections are found at the end of the document.



ERD/LSP-91-0001

Figure 1-1. Major components of the risk assessment process.

Section 1 References

U.S. Environmental Protection Agency (U.S. EPA) (1986), *Superfund Public Health Evaluation Manual,* U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C. (EPA/540/1-86/060, OSWER Directive 9285.4-1).

U.S. Environmental Protection Agency (U.S. EPA) (1988a), *Guidance for Conducting Remedial Investigations and Feasibility Studies under CERCLA,* U.S. Environmental Protection Agency, Office of Remedial Response, Washington, D.C. (EPA/540/1-88/001, OSWER Directive 9355.3-01).

U.S. Environmental Protection Agency (U.S. EPA) (1988b), *Superfund Exposure Assessment Manual,* U.S. Environmental Protection Agency, Office of Remedial Response, Washington, D.C. (EPA/540/1-88/001, OSWER Directive 9355.3-01).

U.S. Environmental Protection Agency (U.S. EPA) (1989), *Risk Assessment Guidance for Superfund. Volume 1. Human Health Evaluation Manual (Part A) Interim final,* U.S. Environmental Protection Agency, Washington, D.C. (EPA/540/1-89/002).

2. Overview of Soil and Ground Water Investigations

D. W. Rice, Jr., and D. W. Layton

2.1. Objective

A key objective of the RI study is to characterize the nature and extent of soil, sediment, and ground water contamination as well as the sources* of that contamination. To that end, we have installed over 250 monitor wells at the Livermore site and adjacent areas since 1983 and have analyzed thousands of well water samples for various organic, inorganic, and radioactive constituents. Additionally, about 200 boreholes have been drilled from which unsaturated sediment (soil) samples have been similarly analyzed. Results of these analyses show that regions of ground water beneath the site are contaminated, primarily with VOCs such as TCE, PCE, chloroform, and in one location, gasoline and associated components such as benzene, toluene, and xylenes [see Mallon (1989) and also Appendix Q of the RI report (Thorpe *et al.*, 1990) for information on the physicochemical properties of these and other VOCs]. Some naturally occurring inorganic constituents, (e.g., nitrate and sulfate) also occur at elevated levels in several wells. Tritium has been detected at concentrations greater than drinking water standards in two wells onsite. Residual soil and sediment contamination is not widespread, and, where VOCs have been detected, they are typically at trace levels.

In the following subsections, we:

- Provide an overview of previous studies conducted as part of our site investigations.
- Identify the major project areas of interest.
- Discuss the sampling and analysis protocols that have been adopted.

2.2. Background

Hydrogeologic investigations of the Livermore site began in 1980 when 39 test boreholes were drilled in conjunction with a seismic hazards assessment. Ground water monitor wells were constructed in 19 of the test boreholes (Carpenter *et al.*, 1982). Seven of these early monitor wells were sampled and analyzed for tritium, total organic carbon (TOC), total dissolved solids (TDS), and trace metals. Two of the wells were analyzed for VOCs, but none were detected (Stone *et al.*, 1982). Despite the negative results, other evidence evaluated at that time suggested that fuel hydrocarbons near the site of former Building 403 and chlorinated hydrocarbon solvents at various locations at the Livermore site, were potential contaminants requiring further investigation. In 1983, 13 onsite monitor wells were sampled and analyzed for VOCs (Stone and Ruggieri, 1983). Selected ground water samples were also analyzed for major inorganic compounds, TOC, TDS, trace metals, tritium, and polychlorinated biphenyls (PCBs). In

* Throughout the BPHA we use the term "source" for any existing contamination in the environment, with an emphasis on high concentrations of contaminants in media (i.e., sediments and ground water) near sites of past releases. There are no continuing releases to the surface from current activities at LLNL.

addition, aquifer tests were conducted in 10 wells. Sampling results showed several locations with ground water VOC concentrations ranging from 1 to 120 mg/L (equivalent to parts per billion, ppb). The predominant VOC reported in onsite ground water was TCE.

An offsite ground water investigation was conducted in late 1983. Twenty offsite wells were sampled and analyzed for VOCs (Carpenter, 1984). Ground water from four of these wells contained VOC concentrations that exceeded EPA and State guidelines for chlorinated organic compounds in drinking water. The predominant VOC reported in offsite ground water was PCE. A concentration of about 300 mg/L of PCE was detected in a private well a quarter of a mile west of the Livermore site. In this case and subsequently, when VOCs were detected in privately owned wells, LLNL supplied the residents with bottled water and then connected the plumbing of affected residences to the local municipal water supply system and closed the wells.

Four new monitor wells were constructed onsite during 1984, and 108 boreholes were drilled and sampled. A total of 537 sediment samples were analyzed for VOCs, and 43 sediment samples were analyzed for fuel hydrocarbons. Ground water samples were collected from monitor wells and private wells (Carpenter, 1984). The major findings of this investigation included:

- The maximum concentration of VOCs detected in unsaturated sediments was 5.7 mg/kg (equivalent to parts per million, ppm) TCE in a sample from 9.7 m in depth in a borehole south of Building 518.
- Gasoline was detected in sediments from a depth of about 6.1 m to the water table in a borehole in the vicinity of the gasoline station at former Building 403.
- VOCs in ground water appeared to be limited to the two uppermost water-bearing zones below the Livermore site.
- Some private wells existing in 1984 west of the Livermore site might have acted as conduits for downward migration of VOCs.
- No apparent source of PCE for the offsite plume was identified.

During 1985, LLNL compiled data regarding past and present uses, storage, and disposal of hazardous materials by LLNL and previous occupants of the site (Dreicer, 1985). This report included a review of the site history from 1942 to 1985. A substantial correlation was established between areas and types of sediment and ground water contamination, and activities carried out during U.S. Navy occupancy of the Livermore site. Overlay maps were compiled showing the locations through time of underground storage tanks, drum storage racks, sanitary and storm sewers, and temporary waste storage areas. Approximate locations of known and suspected hazardous leaks and spills were identified.

The current monitor well drilling program and comprehensive investigation began in 1985 upon submittal of the LLNL Ground Water Investigation Work Plan Phase II (December 1984) to the RWQCB, DHS, and EPA. The locations of existing monitor wells are shown in Figure 2-1*. A more complete description of the investigations of the Livermore Site Ground Water Project appears in the LLNL Ground Water Project Work Plan (Webster-Scholten and Hall, 1989).

* Throughout this report, contour intervals on figures are in feet as given on figures from previous ground water reports.

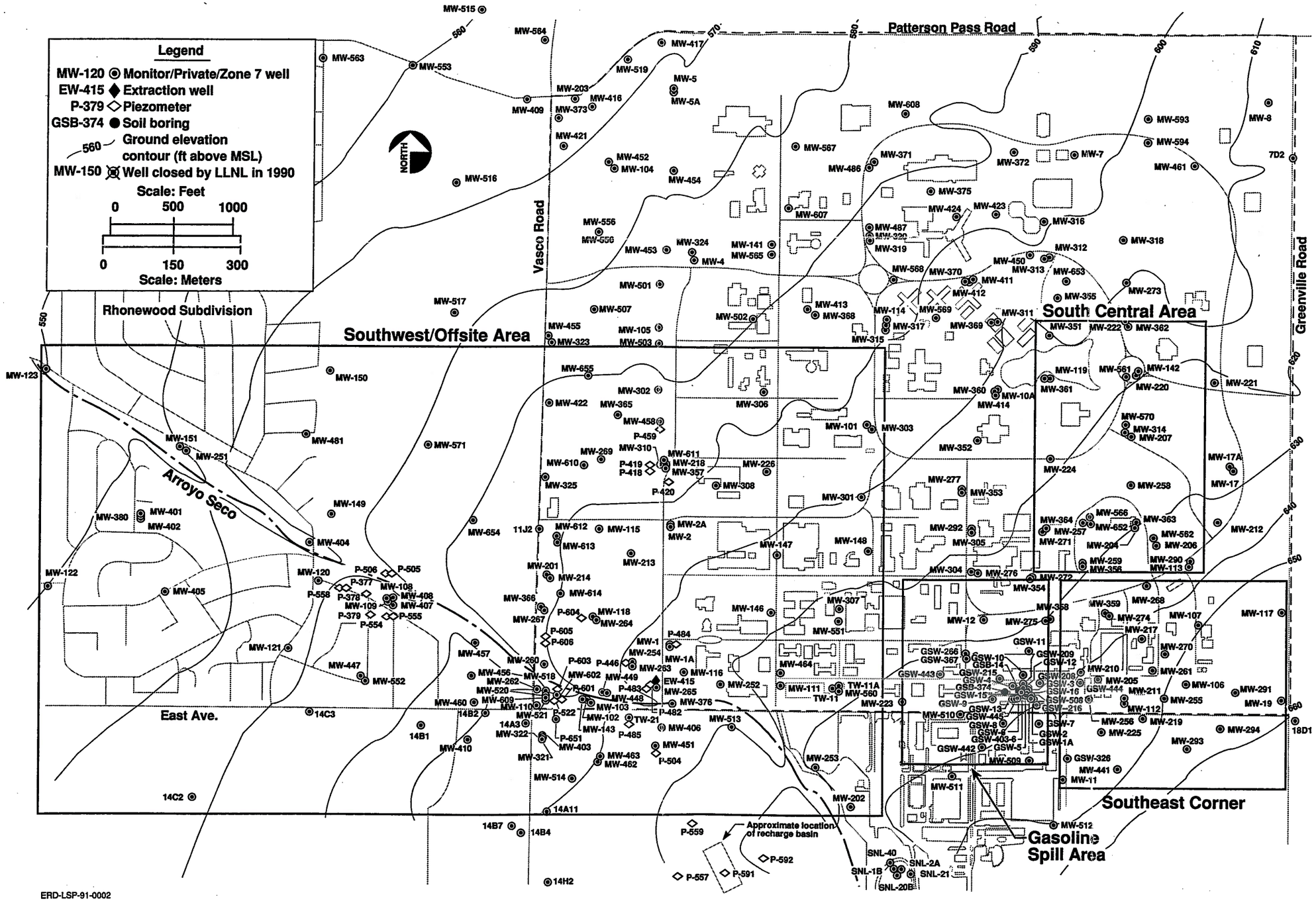


Figure 2-1. Locations of monitor wells at the LLNL Livermore site.

2.3. Project Areas of Investigation

During the assessment of locations at the Livermore site where contaminant releases were likely to have occurred, four areas were identified as potential source locations requiring investigation under DOE Order No. 5480.14 and RWQCB Site Cleanup Order 87-108. The four areas, as shown in Figure 2-2, are:

- Southwest section of the Livermore site and the offsite area to the west.
- Gasoline leak site near the former Building 403.
- Southeast section.
- South central section.

To address investigations of the entire site, the RWQCB Site Cleanup Order included a fifth area, the Remainder of Site. These areas are now combined in a single, operable Remedial Investigation/Feasibility Study (RI/FS) unit for CERCLA compliance.

2.3.1. Southwest Quadrant and Offsite

PCE, TCE, 1,1,1,-trichloroethane (1,1,1-TCA), 1,1-dichloroethylene (1,1-DCE), 1,1-dichloroethane (1,1-DCA), carbon tetrachloride, and chloroform have been detected in monitor wells in the southwest quadrant of the Livermore site. A plume of VOCs in ground water extends west of the site, and its limit is defined by offsite monitor wells installed on easements for city streets or on private properties. A portion of the "offsite" plume underlies lands west of the original Livermore site that were acquired by DOE in 1987 as part of a security buffer zone. We are also continuing to investigate low concentrations of TCE in ground water found near the intersection of Vasco Road and Patterson Pass Road (Fig. 2-2). The distribution of VOCs in ground water west of the Livermore site is summarized in Dresen and Hoffman (1986) and updated in Dresen *et al.* (1987).

The former Building 403 (see Fig. 2-3) at the Livermore site was a garage and automotive fuel dispensing facility, first for the Navy and subsequently for LLNL. The area is under investigation as a result of the inventory discrepancy of about 17,000 gal of leaded gasoline accrued over an unknown period prior to 1980. The most credible explanation was failure of the underground tanks, which had been in use since 1942. Sediment sample analyses confirm a release of fuel hydrocarbons to the ground. These tanks were taken out of service in 1979 and filled with sand in 1980. Based on sediment sample analyses, fuel hydrocarbons are present in a column roughly cylindrical in shape and centered under the west end of the southernmost tank. This column is about 12 m in diameter, and extends from a depth of 6.1 m to about 40 m below grade, which is approximately 11 m below the present ground water table. Chemical and hydraulic data suggest very slow movement of fuel hydrocarbons in ground water beneath the spill site. Subsurface distribution of hydrocarbons in the gasoline spill area is summarized in Dresen *et al.* (1986) and Nichols *et al.* (1988) and updated in Thorpe *et al.* (1990).

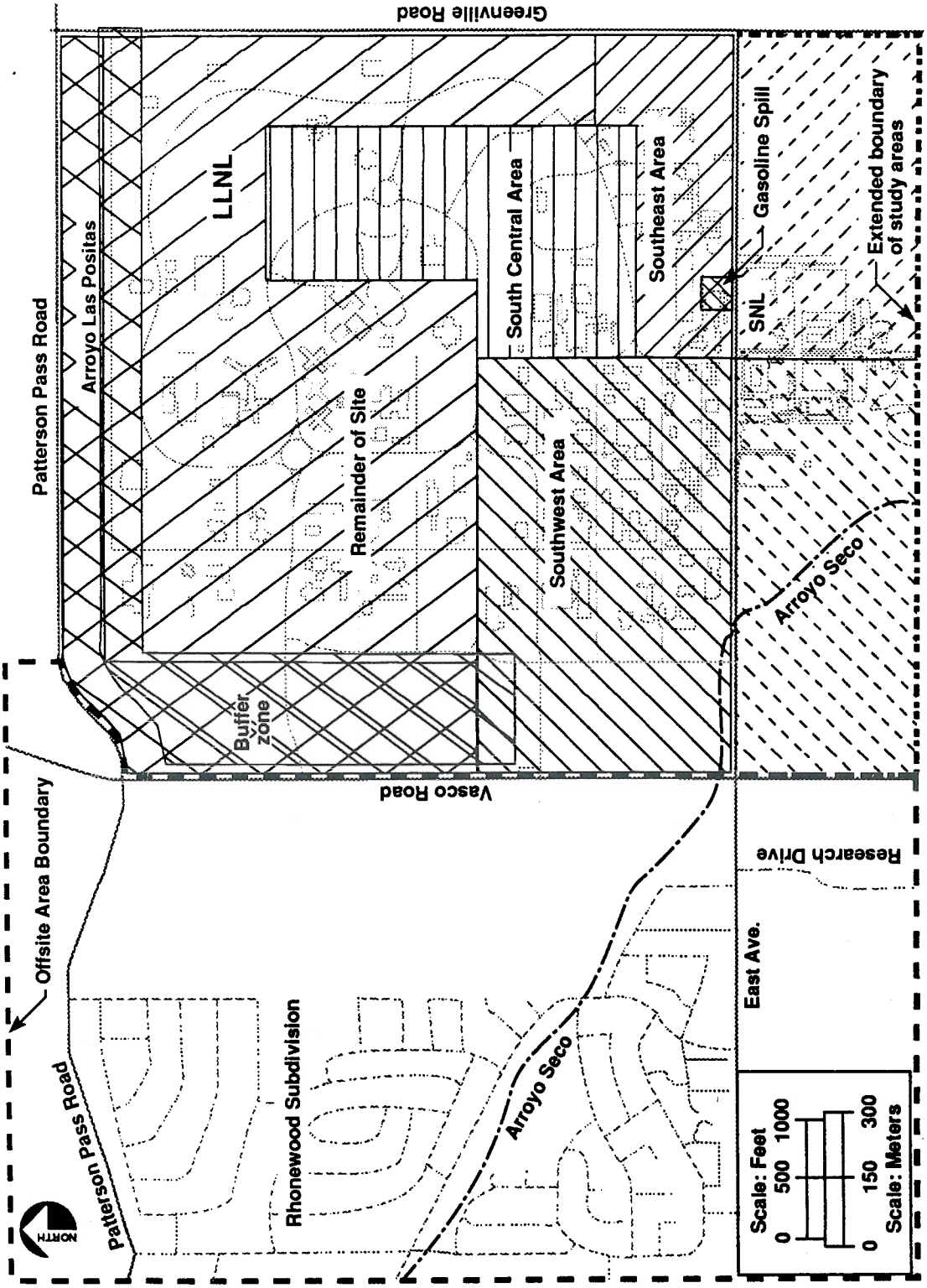


Figure 2-2. The LLNL site environmental remediation study areas.

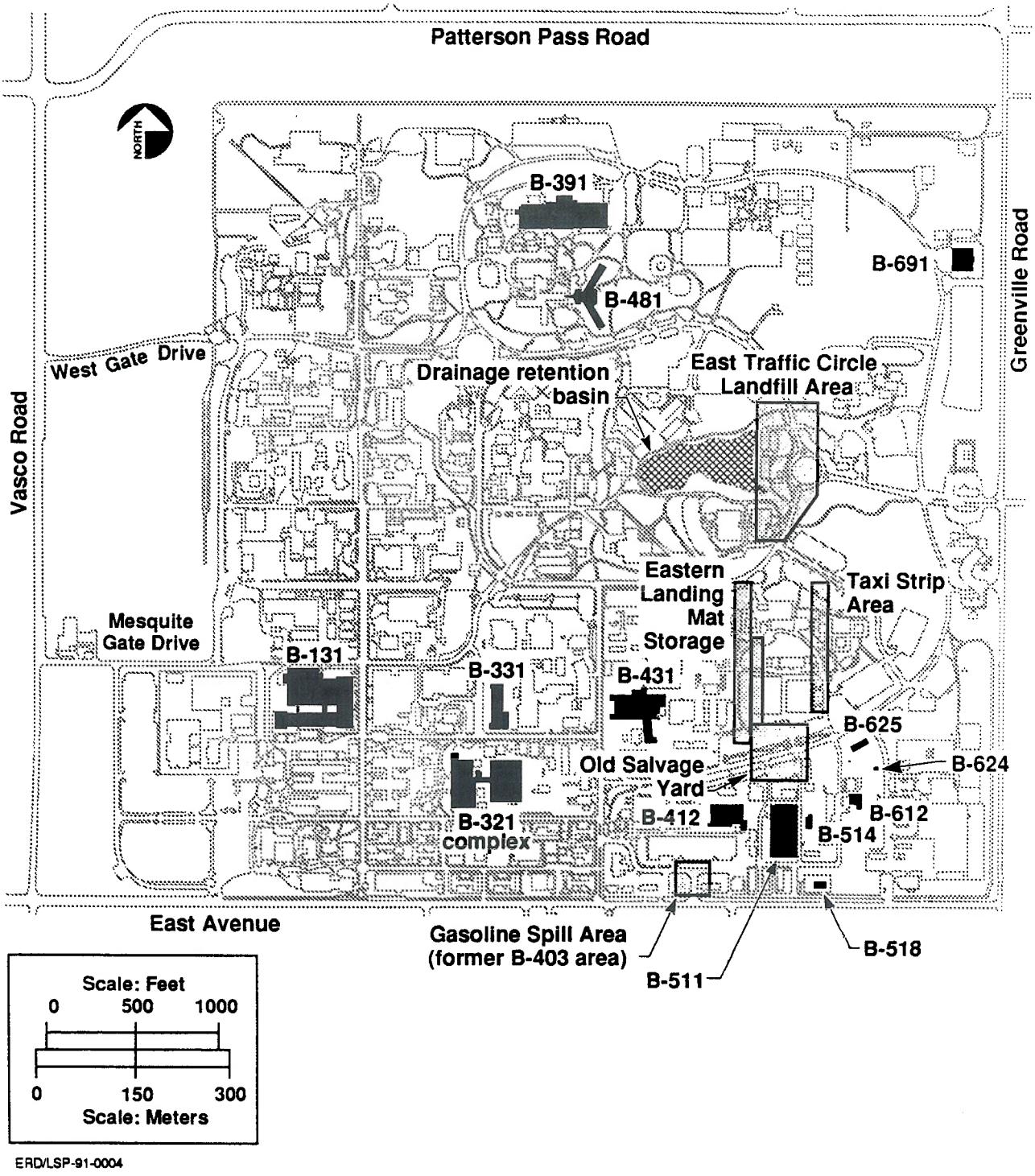


Figure 2-3. Areas or buildings of special interest (adapted from Webster-Scholten and Hall, 1989).

2.3.2. Southeast Section

The southeast section of the Livermore site is under investigation because of the presence of TCE in sediment and ground water samples collected near Building 518 (Fig. 2-3). Navy aircraft engine rework occurred in this area, and beginning in 1959, drums containing solvents were stored there. Sediment sampling at this location was prompted by the poor condition of solvent racks in the area and because large quantities of solvents were used at this building while previously occupied by the Navy. Concentrations of TCE in sediment samples collected on the south side of Building 518 were found to be as high as 5.7 mg/kg (Carpenter, 1984). The distribution of VOCs in the ground water in the southeast area and vicinity is summarized in Dresen and Nichols (1986) and Thorpe *et al.* (1990).

2.3.3. South Central Section

A considerable amount of cleanup has occurred in the south central section of the Livermore site. Complete summaries of the earlier remediation efforts at the Taxi Strip Area (waste storage) and the East Traffic Circle Landfill Area are presented in Buerer (1983) and McConachie *et al.* (1986), respectively (see Fig. 2-3). During the 1982 initial sampling and low-level radiological survey of the Taxi Strip Area within the south central portion of the site, several small waste-disposal pits, associated with solar evaporation trays used for waste volume reduction, were found. The pits were excavated to varying depths (the deepest was 10 m), depending on the extent of the sediment contamination found. Approximately 3,000 m^3 of sediment was removed from the Taxi Strip Area. Sediment showing radioactivity above uncontrolled area limits was transported to a DOE disposal site. The remainder of the excavated sediment was disposed of at a State of California certified hazardous waste facility.

In 1984, the East Traffic Circle Landfill, an inactive landfill located in the south central portion of the Livermore site, was uncovered by construction workers. Aerial photographs taken during the mid-1940s do not show the landfill, although it does appear in LLNL photographs from 1956 as a large depression with a road to the bottom. The landfill was apparently in use until about 1970 when the area was returned to grade. Record searches have been performed to determine the contents and boundaries of the landfill. Activities conducted in the area may have included:

- Burial of construction and metal debris.
- Disposal of capacitors, some containing PCBs.
- Disposal of various drums, some of which may have contained chemical and low-level radioactive wastes.
- Disposal of bright dip (plating) tank contents.
- Storage of hydrocarbon fuel in aboveground tanks.
- Disposal of sandblasting sand, grass cuttings, and gardening debris.

The boundaries of the landfill were verified by careful trenching with a backhoe. Soil and sediment sampling was conducted to define the areas associated with different disposal activities. Shallow soils generally showed less than 0.1 mg/kg volatile halogenated and nonhalogenated organic chemicals. However, a sample collected beneath 20 partially crushed metal drums

showed 11 mg/kg TCE and 50 mg/kg PCE. Sediments 6.4 to 15.5 m beneath the drums showed TCE and PCE concentrations approaching 1 mg/kg each. During 1984, the East Traffic Circle Landfill Area was excavated to a depth of 1.5 to 2 m below grade and to 10 or more m in the vicinity of the capacitors. Almost 11,000 m^3 of soil and debris were excavated and disposed of at State-permitted hazardous waste disposal sites.

Nonetheless, residual VOCs in deep unsaturated sediments in the South Central Area remain a potential source of VOCs in ground water. During 1986, water samples from monitor well MW-206, southeast of Building 543, contained 5,800 mg/L of TCE, one of the highest concentrations observed to date in ground water at the Livermore site. During 1987, TCE concentrations in water samples from MW-206 had declined to about 2,500 mg/L. In addition, this well also contained 40,000 picocuries per liter (pCi/L) tritium when measured in 1985; this amount is twice the 20,000 pCi/L that corresponds to State and Federal standards for drinking water (DHS,1988; U.S. EPA, 1988). However, tritium has a relatively short half-life of 12.3 y and its low energy [18 kilovolts (kV) maximum beta particle emission] would result in a low annual radiation dose if this well were used for drinking water. Repeat sampling of MW-206 during 1988 showed a decrease in tritium to 28,000 pCi/L, and repeat sampling during 1989 showed a further decrease in tritium to 25,000 pCi/L [see Table 4.2.3-13 in the RI report (Thorpe *et al.*, 1990)].

2.3.4. Remainder of Site

As defined in RWQCB Site Cleanup Order 87-108, the Remainder of Site Area is an irregular parcel encompassing the localities not covered in other areas of the Livermore site. Potential releases in this area are based on reports of past activities related to oil spilled or drained from drums, flammable materials burned in fire training activities, spills to the ground from dip tanks, storage of radioactive material, three spills of radioactive material near Building 251 (cleanup has been completed), burial of noncontaminated animals used for biomedical research, the use of garbage pits, and a research project on plutonium uptake and resuspension from a garden using sanitary sewage sludge. Details of these activities, areas involved, and quantities of materials used are presented in Dreicer (1985), Webster-Scholten *et al.* (1987), and Thorpe *et al.* (1990).

2.4. Ground Water, Sediment, and Soil Investigations

Ground water, sediment, and soil data are required to:

- Assess the lateral and vertical extent of contamination on and off the Livermore site.
- Understand the hydrogeologic characteristics under the Livermore site and adjacent affected areas.
- Determine the nature and location of possible sources of contamination.
- Develop public health assessments.
- Evaluate potential remedial action alternatives and engineering designs.
- Characterize baseline conditions.

The types of data required are varied and often interrelated. The primary investigative techniques used to define the lateral and vertical extent of VOCs in ground water, sediment, and soils, and to locate VOC release sites are:

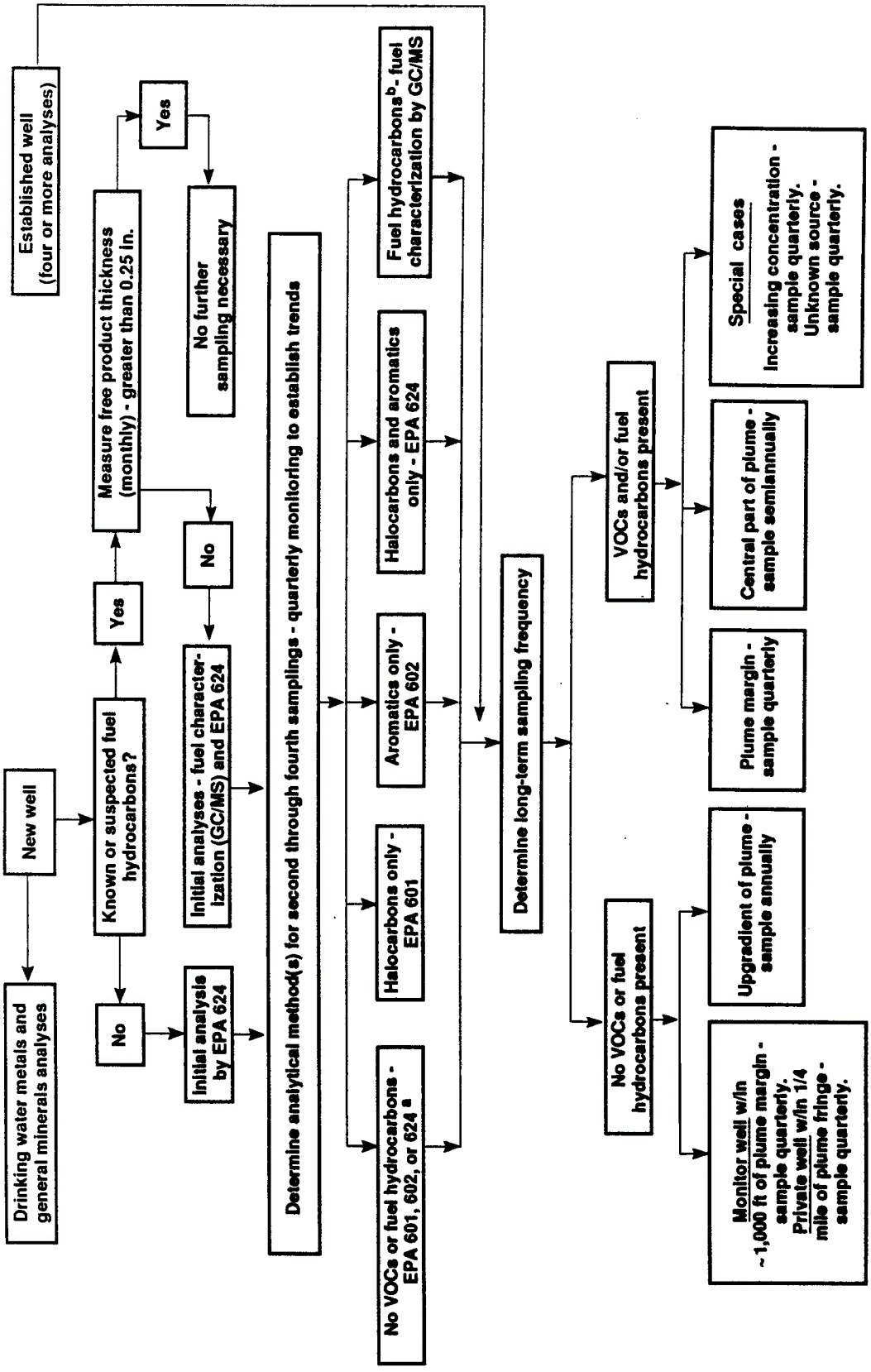
- Monitor well installation.
- Surficial soil, saturated and unsaturated sediment, and ground water sampling.
- Chemical analyses.

The primary investigative techniques used to understand the hydrogeologic characteristics of the area include:

- Development of a three-dimensional representation of subsurface geology derived from hydrogeologic studies of the area.
- Determination of aquifer characteristics by conducting pumping and slug tests.
- Determination of water table configuration. Analytic and numerical contaminant transport models are used to aid in source definition, evaluation of remedial alternatives, and risk assessment.

Descriptions of drilling and well construction conducted prior to 1985 are presented in Stone *et al.* (1982) and Carpenter (1984). Since 1985, well construction has been under the supervision of Weiss Associates, Oakland, California. Techniques for borehole drilling and logging, sediment sampling and analysis, well design and construction, and well development for this group of wells are described in Rice and Daley (1988). For each well constructed, lithologic and geophysical logs are prepared that include natural gamma, point resistivity, and 6-ft (2-m) lateral resistivity. The protocol for sampling and analyzing well waters is depicted in Figure 2-4. The initial samples of ground water from each new monitor well are analyzed for both organic and inorganic constituents. Organic constituents are analyzed by gas chromatography/mass spectrometry (GC/MS) (EPA Method 624) to screen them for Target Compound List (TCL) pollutants and to identify any nonquantifiable compounds (e.g., aliphatics). These data are then evaluated to determine the appropriate EPA method of analyses for subsequent samples, which is generally GC purge and trap (EPA Method 601). Regular ground water sampling of monitor wells provides data on plume movement, remediation effectiveness, and physical factors that may influence measured ground water concentrations. We also make baseline field measurements of the temperature, pH, and conductivity of ground water.

The current protocol for sampling and analyzing soils and unsaturated sediments is depicted in Figure 2-5. Sediment samples are collected during drilling operations to determine changes in VOC concentrations with depth and in source investigations to determine near-surface concentrations of contaminants. In addition, samples are taken of surficial soils and sediments to characterize the potential migration of organic, inorganic, and radioactive substances from source areas. Soil and sediment samples are also taken at fixed sampling locations at the Livermore site and offsite areas as part of the ongoing environmental monitoring program conducted to monitor levels of various radioactive and nonradioactive substances in environmental media.



a EPA 601 analyses used if nearest wells contain halocarbons only; EPA 602 if aromatics only; or EPA 624 if halocarbons, aromatics, or aliphatics.

b Fuel hydrocarbons (C_4 to C_{12}), including benzene, ethyl benzene, toluene, xylene isomers, aliphatic and alicyclic hydrocarbons, and ethylene dibromide.

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Figure 2-4. Flowchart for LLNL ground water sampling plan.

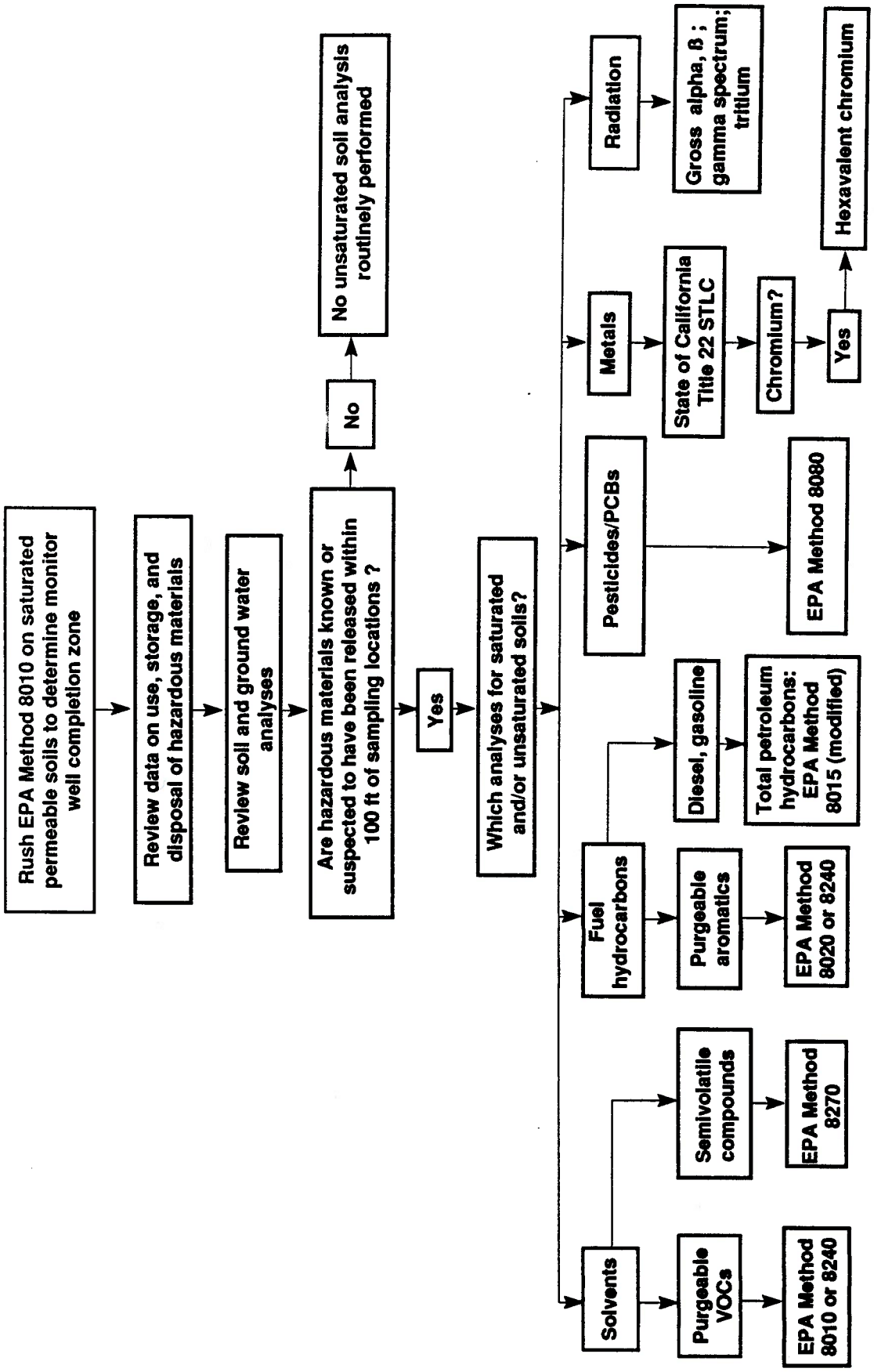

Figure 2-5. Flowchart for LLNL soil sampling plan.

2.5. Surface Water Monitoring

No perennial surface water exists in the vicinity of the Livermore site. Intermittent surface water at the site is comprised of storm water runoff and treated effluents from LLNL's ground water investigations discharged to the storm sewers. Some surface water is directed via storm channels into an excavated 1.6 hectare (ha) retention basin in the central portion of the Livermore site. Runoff from about 356 ha of a 405-ha watershed also flows into this basin when precipitation is greater than approximately 1 cm/day with dry initial field conditions. The basin has a 34,000 m^3 storage capacity and is a significant source of ground water recharge (Toney, 1988). Other surface runoff at the Livermore site flows into storm channels, eventually reaching the Arroyo Seco or Arroyo Las Positas, or it flows directly into these arroyos, bypassing the feeder routes. Except during a few storm events each year, discharged waters infiltrate quickly into soils and do not leave the site as runoff.

Surface waters caused by storm events at six locations on and near the Livermore site, including the storm channels, arroyos, and the retention basin, are analyzed for VOCs, base/neutral/acid extractable compounds, radioactivity, and a number of metals. Details of sampling activities and chemical analyses are presented in annual LLNL Environmental Protection Department Monitoring Reports [e.g., Holland and Brekke (1988)]. Treated effluent from the ground water investigation is discharged only when it meets specified effluent limitations set forth in the current Waste Discharge Orders issued by the RWQCB. These limits are at or below State of California drinking water standards. Any traces of VOCs that might reach surface waters will rapidly volatilize.

2.6. Air Monitoring

Air quality is monitored at the Livermore site perimeter and offsite locations for airborne radionuclides and beryllium. Details of monitoring activities and subsequent results are presented in annual LLNL Environmental Protection Department Monitoring Reports [e.g., Holland and Brekke (1988)].

Volatilization of VOCs from the vadose zone (i.e., unsaturated zone) or even the ground water is theoretically a slow, ongoing process. Previous VOC releases to the ground at the Livermore site have aged, and decades of volatilization would have released most of the available residuals; thus, measurable levels in the air are not expected. Regular monitoring to measure such outgassing is impractical because the general background of VOCs from permitted air discharges in the industrial setting of the Livermore site would mask the low concentrations resulting from outgassing from soil or sediments. General mixing of air at the surface and natural degradation of the halogenated organic solvents in sunlight further minimizes concern over air contamination from solvents that have outgassed. Even though the exhalation of VOCs from soils to air is expected to be minimal and is difficult to measure, it does represent a potential exposure pathway, and consequently, we do assess this pathway using estimates of the volatilization rate derived from a transport model.

Air monitoring for emissions resulting from well sampling and other activities of the LLNL ground water investigations has not been deemed necessary by the Bay Area Air Quality Management District (BAAQMD), owing to the low emissions involved, and therefore, is not

currently included in the environmental restoration effort. Pilot ground water remediation studies are being conducted using an ultraviolet light/hydrogen peroxide system, which produces no air emissions. Nevertheless, these pilot studies are being conducted under permits from the BAAQMD.

2.7. Chemical Analyses and Quality Assurance

All analyses of organic compounds have been performed by contract analytical laboratories (CALs) certified by the State of California since 1984. Between 1985 and 1988, several special quality control experiments were performed to evaluate sources of variability in the chemical analyses of VOCs in ground water beneath the Livermore site (McConachie *et al.*, 1988). Beginning in March 1988, we implemented the additional Quality Assurance/Quality Control procedures described by Rice (1988), such as monthly QC reports from CALs and the use of controlled field logbooks. Prior to 1988, analyses for radioactivity were performed either by a CAL or by LLNL. Since January 1988, all analyses for radioactivity have been performed by a CAL. Objectives for precision, accuracy, and completeness are based on the previous analytical performance of CALs used by the LLNL Ground Water Project. The specific procedures used to assess data precision, accuracy, and completeness are summarized in the Quality Assurance Program Plan (Rice, 1988).

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3. Contaminant Concentrations in Environmental Media and Potential Exposure Pathways

D. W. Layton, J. I. Daniels, and V. M. Johnson

The emphasis of the risk assessment is on human exposure (i.e., public health). In this section, we demonstrate that the only important migration pathway for contaminants is via the ground water. The discussion is divided into two parts:

1. Section 3.1 describes the potential routes of contaminant migration and exposure. This provides the background for Section 3.2, which analyzes each pathway.
2. Section 3.2 is a screening analysis, from which we conclude that VOCs in the ground water are the contaminants of interest.

3.1. Potential Routes of Migration

Figure 3-1 is a conceptual diagram of the potential exposure media and transport pathways for the LLNL site. At potential source regions where VOCs, for example, may occur in surficial soils, the possible exposure pathways consist of dermal contact and inhalation of compounds that are volatilized from soils or emitted via the resuspension of contaminated soils. Because access to LLNL is strictly controlled for security reasons, the population presently at risk in this case consists of workers who may periodically come into contact with such soils. For the baseline assessment of the public health risk, we assume that the LLNL site will remain as a research and development facility owned and actively funded by the DOE.

According to the Federal Facility Agreement (U.S. EPA *et al.*, 1988), no change of ownership can occur without implementation of specific provisions for various response actions (e.g., treatment systems and monitoring systems). Moreover, the basic nature of the site is unlikely to change—even with a change in ownership—because of the valuable office and laboratory facilities, along with roads and utilities, located across the site. For present and future offsite populations, the possible exposure pathways consist of (1) contact with surface water runoff or sediment in local arroyos that receive drainage waters from the site and (2) the use of contaminated well waters for drinking and bathing.

We estimate contaminant concentrations in the different media by the use of transport and fate models (e.g., simulation of the movement and degradation of a contaminant in ground water) or direct measurements (e.g., measured concentration of a contaminant in soil or sediment). Human exposures are then calculated by constructing alternative scenarios leading to contact with contaminated media. Occupational scenarios focus primarily on the frequency and duration of contacts with contaminants at source regions; whereas for the nearby public, we focus on lifetime exposures to contaminants in well waters and transient exposures to sediments and surface runoff. To address uncertainties involved in our analyses, we adopt "health-conservative" and "best-estimate" scenarios and assumptions that yield estimates of risk, which represent a range of plausible predictions.

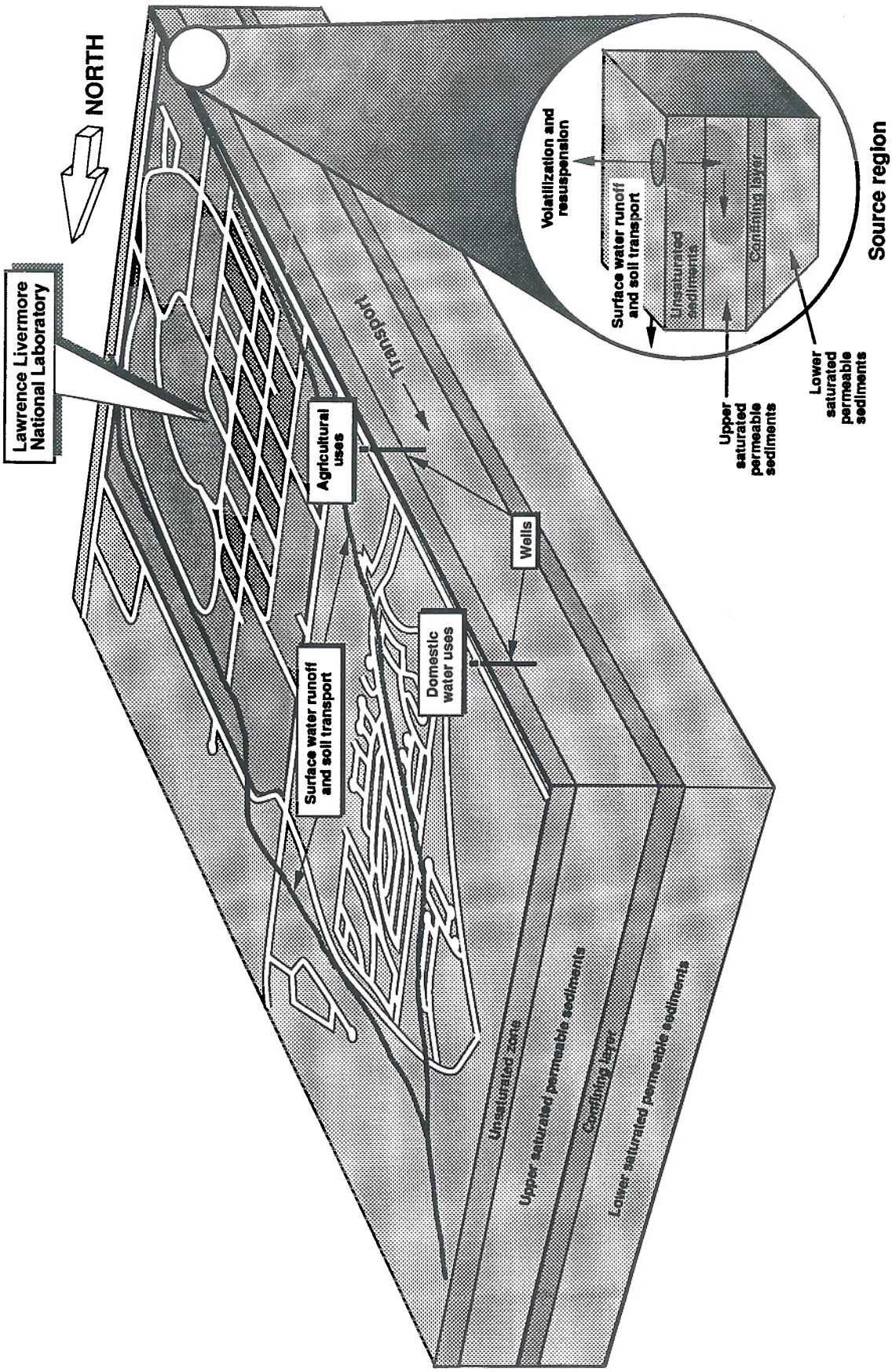

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Figure 3-1. Conceptual diagram of contaminant transport pathways and potential exposure media for the LLNL site.

3.2. Contaminant Persistence

From the standpoint of public health, the most important contaminants detected in the soils and ground waters in the vicinity of the LLNL site are those substances that pose the greatest health risks to people living near the site. The primary factors determining the potential risk of a contaminant are the nature and magnitude of potential human exposures and the contaminant's toxicity (e.g., carcinogenic potency). Potential exposures are governed by the persistence of media contamination and the ways that populations at risk could come into contact with contaminated media. With these considerations in mind, we developed a screening methodology designed to categorize the most important contaminants at the LLNL site. This methodology is based on (1) the occurrence and concentrations of those contaminants in environmental media, (2) comparisons of their measured concentrations with applicable concentration limits defined by Federal and State regulations, and (3) their relative toxicities.

3.2.1. Screening Methodology

Our conceptual model of the potential exposure pathways to contaminants released from previous operations at the LLNL site indicates that releases occurred at or near the soil surface. These releases were followed by gravity flow and/or leaching of the contaminants through the unsaturated zone to ground water. We conducted a detailed program of sampling and analysis in the vadose zones beneath the known surficial sources to describe this process. Soil-based releases to the atmosphere occur via volatilization of gases and resuspension of particles. Soil-based releases to surface waters occur via surface runoff and erosion of contaminated soil/sediment. Contaminants in soil/sediment may migrate to ground water or remain sorbed until degradation to nontoxic residuals. Based on this conceptual model, the contaminants detected in well waters should correspond reasonably well with those detected in soil because ground water contaminants were originally derived from releases to surface soils. However, exceptions are organic substances that are strongly sorbed to soils and sediments and leach to ground water at a slow rate, and degradation products resulting from biotransformation of original contaminants.

Our initial screening analyses focused on the identification of those ground water contaminants that have the highest exposure potential and toxicity. After primary ground water contaminants were identified, we reviewed data on soil/sediment contaminants to confirm that the ground water contaminants of concern do, in fact, correspond with the primary soil/sediment contaminants. We also determined whether other contaminants occurred in soils that should be considered in our health-risk assessments.

3.2.2. Hazardous Substances in Ground Water

Figure 3-2 shows our screening methodology for ground water contaminants. We began our evaluation of the exposure potential of the organic, inorganic, and radioactive substances detected in ground waters in the vicinity of the LLNL site by categorizing substances according to the frequency with which they have been detected. The data for organic and inorganic chemicals detected in ground water samples that are used for this and other analyses to be described in this section appear in the RI report (Thorpe *et al.*, 1990, Appendix M). We used three categories for this initial phase of the screening process:

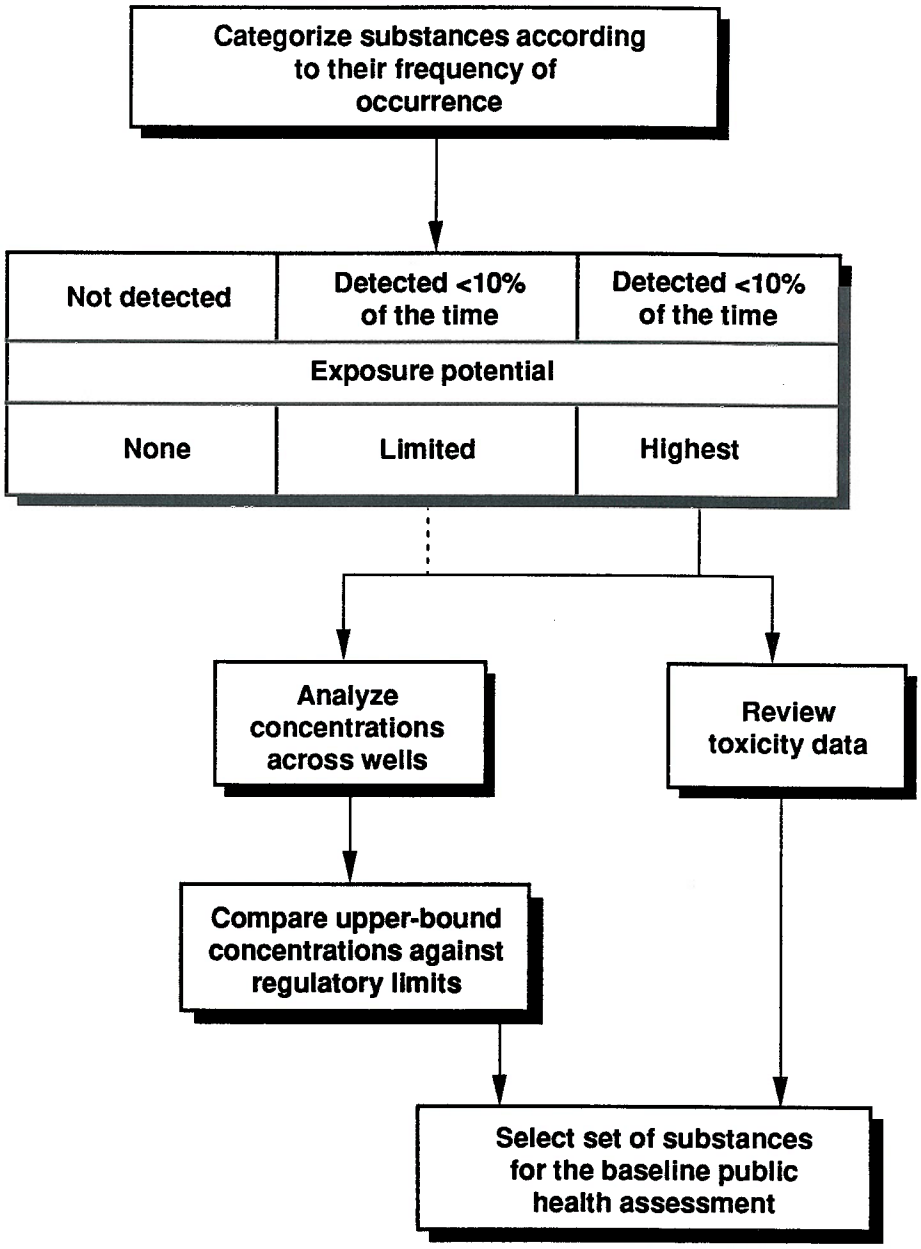

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Figure 3-2. Methodology for selecting the substances to be addressed in the public health assessment.

1. Substances looked for but not detected.
2. Substances detected less than 10% of the time.
3. Substances detected 10% or more of the time.

We selected the first two categories to separate those substances with the least exposure potential (detection frequency <10%) from substances with the greatest exposure potential (detection frequency ≥10%). Detection frequencies were calculated by dividing the number of samples with detectable concentrations of a substance by the total number of water samples taken for the specific purpose of detection. The detection frequency of 10% was used simply to group the various constituents of ground water. Even though our primary focus was on the substances detected greater than 10% of the time, we evaluated the potential significance of the substances that occur less frequently. The total number of applicable water samples varied for each substance. This number depended on the number of wells sampled to determine the presence of the substance and the number of multiple samples (i.e., time-series samples) drawn from individual wells. The concentration data we evaluated were collected over a 68-month period beginning March 1983 and ending October 1988. This data cut-off date was necessitated by the time required for a comprehensive analysis. However, when data in that set left a question that could be resolved by subsequent data, the later data were considered.

After the frequency of detection data were used to group the substances, we compared the concentrations for the various substances with applicable regulatory limits. We then used data on the concentrations of the various substances along with hydrogeologic data on the thicknesses of permeable sediments containing the contaminants to determine the total masses of the principal constituents dissolved in ground water. The final selection of the substances used to assess the public-health risks of ground water contamination at the site was based on the frequency-of-detection data, concentrations in ground water, total masses of the principal constituents in ground water, and toxicity data.

Table 3-1 lists 75 constituents of ground water that were looked for, but were not detected, in water samples drawn from monitor wells. Included are compounds such as polychlorinated biphenyls (PCBs), polycyclic aromatic compounds, pesticides, plasticizers, and vinyl chloride. These compounds have not been associated with operations constituting potential sources of soil and ground water contamination at the LLNL site. Thus, it is not surprising that detectable concentrations were not measured. Along with the chemical measurements made on water samples, 145 water samples were tested for the presence of fecal coliform. Only one sample yielded a positive result. Table 3-2 lists an additional 33 organic, inorganic, and radioactive constituents that were detected at least once, but less than 10% of the time. We note that some of the samples showing detection are from wells that have not indicated the presence of the constituent in subsequent sampling.

Table 3-3 lists those constituents of ground water that were detected greater than or equal to 10% of the time in water samples. As expected, many inorganic substances appear in this table because they are natural components of sediments and ground water. The organic compounds that were detected frequently were primarily chlorinated solvents (e.g., TCE, PCE, and 1,1,1-TCA) and compounds associated with gasoline-contaminated water (e.g., benzene, toluene, and xylenes). Other frequently detected substances include co-contaminants, such as

Table 3-1. Inorganic and organic constituents of ground water looked for, but not detected, in water samples drawn from monitor wells at the LLNL site.

Constituent	Number of samples	Lower limit of detection (μg/L)[a]	Constituent	Number of samples	Lower limit of detection (μg/L)[a]
PCB 1248	5	0.3	2,4-dichlorophenol	90	1
PCB 1016	7	0.3	Diethylphthalate	90	1
PCB 1221	7	0.3	2,4-dinitrotoluene	90	1
PCB 1232	7	0.3	2,6-dinitrotoluene	90	1
PCB 1242	7	0.3	Di-n-octylphthalate	90	1
PCB 1254	7	0.3	1,2-diphenylhydrazine	90	1
PCB 1260	7	0.3	Fluoranthene	90	1
PCB 1262	7	0.3	Fluorene	90	1
Total PCBs	9	0.3	Hexachlorobenzene	90	1
Carbon disulfide	40	1	Hexachlorobutadiene	90	1
2-hexanone	40	1	Hexachlorocyclopentadiene	90	1
Styrene	40	1	Hexachloroethane	90	1
Vinyl acetate	40	1	Isophorone	90	1
Indeno(1,2,3-c,d)pyrene	89	1	Nitrobenzene	90	1
Acenaphthene	90	1	2-nitrophenol	90	1
Acenaphthylene	90	1	Pentachlorophenol	90	1
Anthracene	90	1	1,2,4-trichlorobenzene	90	1
Benzo(a)anthracene	90	1	N-nitrosodiphenylamine	91	1
Benzo(b)fluoranthene	90	1	Butylbenzylphthalate	92	1
Benzo(k)fluoranthene	90	1	Bis(2-ethylhexyl)phthalate	134	10
Benzo(g,h,i)perylene	90	1	3,3'-dichlorobenzidine	143	1
Benzo(a)pyrene	90	1	2,4-D	150	0.5
Bis(2-chloroethoxy)methane	90	1	2,4,5-TP (Silvex)	150	0.05
Bis(2-chloroethyl)ether	90	1	Benzidine	151	40
Bis(2-chloroisopropyl)ether	90	1	Lindane	151	0.05
4-bromophenylphenylether	90	1	Endrin	151	0.01
2-chloronaphthalene	90	1	Methoxychlor	151	0.1
2-chlorophenol	90	1	Toxaphene	151	0.5
4-chlorophenylphenylether	90	1	Dibutylphthalate	154	1
Chrysene	90	1	Thallium	154	1
Dibenzo(a,h)anthracene	90	1	Dimethylphthalate	159	1

Table 3-1. (Continued)

Constituent	Number of samples	Lower limit of detection (μg/L)[a]	Constituent	Number of samples	Lower limit of detection (μg/L)[a]
2,4-dinitrophenol	159	10	1,3-dichloropropene	442	0.1
2-methyl-4,6-dinitrophenol	159	1	1,3-dichlorobenzene	1,544	0.1
4-nitrophenol	159	1	cis-1,3-dichloropropene	1,560	0.1
N-nitrosodimethylamine	159	1	2-chloroethylvinylether	2,003	0.5
N-nitrosodi-n-propylamine	159	1	Vinyl chloride	2,003	0.1
Acrolein	408	1	trans-1,3-dichloropropene	2,004	0.1
Acrylonitrile	408	1			

[a]The lowest limit of detection reported for each compound over the entire sampling period.

Table 3-2. Inorganic, organic, and radioactive constituents of ground water detected at least once, but less than 10% of the time, in water samples drawn from wells at the LLNL site.

Constituent	Number of samples	Number of samples with detectable concentrations	Percent detectable
Inorganic			
Beryllium	160	1	1
Silver	176	8	5
Cadmium	175	9	5
Antimony	154	13	8
Organic			
Bromomethane	2,019	1	<1
Chlorobenzene	1,915	2	<1
Chloromethane	2,019	4	<1
Chloroethane	2,019	2	<1
Bromoform	2,019	2	<1
1,2-dichlorobenzene	1,553	1	<1
1,4-dichlorobenzene	1,556	3	<1
Dichlorodifluoromethane	1,512	1	<1
1,2-dichloropropane	2,004	4	<1
1,1,2,2-tetrachloroethane	2,016	3	<1
1,1,2-trichloroethane	2,019	5	<1
4-chloro-3-methylphenol	90	1	1

Table 3-2. (Continued)

Constituent	Number of samples	Number of samples with detectable concentrations	Percent detectable
Trichlorofluoromethane	2,019	17	1
Dibromochloromethane	2,019	29	1
2,4-dimethylphenol	90	1	1
Phenanthrene	90	1	1
Pyrene	90	1	1
2,4,6-trichlorophenol	90	1	1
Bromodichloromethane	2,019	38	2
Methyl ethyl ketone	45	1	2
Naphthalene	92	3	3
Acetone	49	2	4
Methylene chloride	2,019	72	4
Surfactants	177	12	7
Dichlorotrifluoroethane	26	2	8
1,2-dichloroethane	2,046	166	8
Ethylbenzene	551	44	8
Phenol	93	8	9
Radioactive			
Tritium	181	10	6

Table 3-3. Inorganic, organic, and radioactive constituents of ground water detected greater than or equal to 10% of the time in water samples drawn from wells at the LLNL site.

Constituent	Number of samples	Number of samples with detectable concentrations	Percent detectable
Inorganic			
Nickel	154	16	10
Selenium	177	22	12
Mercury	179	24	13
Iron	183	31	17
Manganese	184	35	19
Arsenic	177	38	21
Lead	193	42	22
Barium	24	6	25

Table 3-3. (Continued)

Constituent	Number of samples	Number of samples with detectable concentrations	Percent detectable
Zinc	177	44	25
Trivalent chromium	60	37	62
Chromium	236	149	63
Copper	176	119	68
Boron	152	131	86
Nitrate (as NO_3)	177	163	92
Hexavalent chromium	61	57	93
Sulfate	177	176	99
Organic			
trans-1,2-dichloroethylene	201	35	17
cis-1,2-dichloroethylene	56	10	18
1,2-dichloroethylene (total)	1,798	294	16
Carbon tetrachloride	1,997	368	18
Ethylene dibromide	71	14	19
Toluene	618	119	19
1,1-dichloroethane	1,997	403	20
1,1,1-trichloroethane	1,997	405	20
Freon 113	1,402	365	26
Benzene	622	176	28
Tetrachloroethylene	2,045	695	34
1,1-dichloroethylene	1,997	811	41
Chloroform	1,997	908	45
Total xylene isomers	259	116	45
Trichloroethylene	2,046	1,143	56
Radioactive			
Gross beta	165	117	71
Gross alpha	165	123	75

impurities in industrial-grade chemicals and environmental degradation products (e.g., 1,1-DCE and 1,1-DCA).

Our next step in the screening procedure was to determine which of the substances in Tables 3-2 and 3-3 are most important from a health-effects standpoint. We began by comparing an upper-bound concentration for the compounds (a concentration with a small probability of being exceeded) with the regulatory limits for drinking water established by the State of California DHS and/or the EPA. To derive upper-bound concentrations for that comparative

analysis, we calculated the median concentration values of each substance for all wells sampled. (The number of wells sampled for each substance varied, depending on the historic sampling protocols adopted.) To be conservative, if the calculated upper-bound concentration did not exceed the regulatory limits for a substance, we used the maximum recorded value.

Many of the analytical results reported for the organic and inorganic compounds contained in Tables 3-2 and 3-3 were below detection limits. Values below the limit of detection (LOD) are referred to as "censored" results. To treat such censored results, we followed the recommendation of Gilbert (1987). Here, we divided each LOD reported for a compound by 2 (to estimate the midpoint concentration between zero and the LOD) and then computed the median concentration from the concentrations above the LOD as well as the calculated midpoint values. Probability plots of the resulting concentration data for the different substances indicate that the concentrations are approximated by lognormal distributions.

Table 3-4 summarizes the maximum concentrations observed, geometric mean (GM), and geometric standard deviation (GSD) for the inorganic and organic constituents detected 10% or more of the time. We also include the total number of wells sampled for each substance and the number of wells yielding water with no detectable concentrations of a substance (i.e., only censored data). For most of the substances, the number of clean wells is significantly larger than the number of wells producing water with detectable concentrations.

3.2.3. Comparison with Regulatory Limits

As one means of determining which of the substances detected are most important from a public-health standpoint, we compared upper-bound concentrations of substances with applicable regulatory limits for drinking water. These regulatory limits are Federal and State primary and secondary drinking water standards and State drinking water action levels. Primary drinking water standards are maximum contaminant levels (MCLs) that are designed to protect against adverse health effects (e.g., cancer or systemic effects). Secondary drinking water standards are MCLs that typically protect against undesirable organoleptic effects (e.g., adverse taste, odor, or color). State drinking water action levels are recommended maximum concentration limits. To minimize the possibility of failing to identify a substance of potential concern, we selected the upper-bound value as the 99th cumulative percentile concentration on the lognormal distribution for each substance. This value is the product of the GM and GSD^Z, where $Z = 2.3$, or the number of standard deviations corresponding to the 99th cumulative percentile on a lognormal distribution. To ensure that a substance was not omitted from consideration, we also compared maximum reported concentrations to the respective Federal and/or State regulatory limits and action levels for drinking water just mentioned.

3.2.3.1. Inorganic Constituents

Our review of the inorganic constituents of ground water begins with an analysis of the concentration data for substances that were detected infrequently. Our goal was to determine if these substances are present at levels above applicable limits. The four inorganic substances that occurred less than 10% of the time were beryllium, silver, cadmium, and antimony (see Table 3-2). Both the State of California (DHS, 1989a) and the U.S. EPA (1988a) have established a primary drinking water standard for silver of 0.05 mg/L and for cadmium of

Table 3-4. Statistical summary of the lognormal statistics of concentration data for inorganic, organic, and radioactive constituents detected with a frequency greater than or equal to 10% in well waters.

Constituent	Total number of wells	Number of wells with only censored data[a]	Maximum concentration[b]	Units	Lognormal statistics GM[c]	Lognormal statistics GSD[d]
Arsenic	160	122	2.7E–02	mg/L	0.00078	2.3
Barium	23	17	1.2E+00	mg/L	0.089	2.7
Benzene	249	221	4.5E+04	µg/L	0.97	10.1
Boron	144	19	1.1E+01	mg/L	0.49	2.5
Carbon tetrachloride	266	192	7.9E+01	µg/L	0.58	3.9
Chloroform	266	99	8.7E+02	µg/L	1.1	5.3
Chromium	164	26	1.0E–01	mg/L	0.012	2.0
cis-1,2-dichloroethylene	34	25	5.0E+01	µg/L	0.75	7.7
1,1-dichloroethane	266	193	5.0E+02	µg/L	0.62	4.1
1,1-dichloroethylene	266	145	6.4E+02	µg/L	1.0	5.8
Copper	161	45	4.6E–02	mg/L	0.0028	2.5
Ethylene dibromide	22	12	5.8E+02	µg/L	0.80	25.9
Freon 113	239	120	2.9E+02	µg/L	0.56	4.1
Gross alpha	150	39	1.6E+01	pCi/L	1.3	2.5
Gross beta	150	44	2.9E+01	pCi/L	1.9	3.9
Hexavalent chromium	31	3	7.8E–02	mg/L	0.013	3.3
Iron	161	134	6.7E+00	mg/L	0.017	2.8
Lead	174	132	2.7E–01	mg/L	0.00083	3.0
Manganese	161	131	1.1E+01	mg/L	0.0082	3.8
Mercury	160	136	9.0E–04	mg/L	0.00006	1.6
Nickel	146	130	9.5E–01	mg/L	0.00066	2.5
Nitrate (as NO_3)	161	13	7.5E+01	mg/L	13	4.3
Selenium	160	140	1.4E–02	mg/L	0.00091	1.7
Sulfate	161	1	1.1E+03	mg/L	41	2.3
Tetrachloroethylene	267	151	1.8E+03	µg/L	1.0	7.8
Toluene	249	211	5.6E+04	µg/L	0.82	6.3
Xylene isomers (total)	68	41	2.4E+04	µg/L	3.2	23.2
1,2-dichloroethylene (total)	266	202	5.0E+02	µg/L	0.55	3.3
trans-1,2-dichloroethylene	107	88	5.0E+01	mg/L	0.45	4.0
1,1,1-trichloroethane	266	176	5.0E+02	µg/L	0.53	3.2
Trichloroethylene	267	99	5.8E+03	µg/L	2.6	13.3
Trivalent chromium	31	8	1.3E–02	mg/L	0.0017	2.6
Zinc	161	119	2.6E–01	mg/L	0.0075	2.3

[a]Concentrations below the LOD are censored data. Remaining wells had concentrations that are either ≥LOD (uncensored data) exclusively or both ≥LOD and <LOD.

[b]Compiled from data contained in Appendix Table M-1 of the RI report (Thorpe *et al.*, 1990).

[c]Geometric mean.

[d]Geometric standard deviation.

0.01 mg/L. No standards are established for beryllium and antimony. Silver was detected in 8 of 164 monitored wells, with one well (MW-406) containing a concentration of dissolved silver as high as 0.08 mg/L. None of the cadmium concentrations exceeded 0.01 mg/L. Beryllium was detected in only one well (11Q6). (This was a private well, no longer in existence, located about 1 km west of the original LLNL site boundary). Here, the measured concentration of beryllium was 0.005 mg/L. The maximum concentration of antimony was 0.006 mg/L for 13 of 146 monitored wells that produced water with detectable antimony concentrations.

Table 3-5 includes our comparisons of the various regulatory limits with the upper-bound and maximum observed concentrations (see Thorpe *et al.*, 1990, Appendix Table M-1) for inorganic substances detected 10% or more of the time. These data indicate that most of the naturally occurring inorganic constituents of ground water occurred at levels below limits of regulatory concern. For example, the calculated upper-bound concentrations (99th cumulative percentile) of arsenic, barium, copper, iron, lead, mercury, selenium, and zinc did not exceed any Federal and State maximum concentration limits.

Although the calculated upper-bound limits did not exceed regulatory limits for these metals, *maximum* concentrations of barium, iron, lead, and selenium in some well waters were above these limits. Barium has a Federal and a State MCL of 1 mg/L (U.S. EPA, 1988a; DHS, 1989a). Barium was detected at concentrations of 1 and 1.2 mg/L in two wells (MW-406 and MW-407) out of 23 tested. However, 17 of the tested wells contained barium below detection limits. We found 27 wells of the 161 wells tested had detectable concentrations of iron. Wells MW-012, MW-411, 5F1, and 18D1 produced water with iron at concentrations ranging from 0.39 to 6.7 mg/L. By comparison, the Federal and State MCLs for iron are both 0.3 mg/L (U.S. EPA, 1988d; DHS, 1989d). Concentrations of lead were below the Federal and State MCLs of 0.05 mg/L (U.S. EPA, 1988a; DHS, 1989a) in 174 wells monitored. However, two other wells (GSW-5 and GSW-403-6) that extended into the saturated zone in the immediate vicinity of the subsurface leak of leaded gasoline contained lead concentrations of 0.16 and 0.27 mg/L, respectively, which exceed regulatory limits. Selenium was detected in 20 of 160 wells tested, but only well MW-002 produced water containing selenium (0.014 mg/L) above the Federal and State MCL of 0.01 mg/L (U.S. EPA, 1988a; DHS, 1989a).

The upper-bound concentrations of total chromium, manganese, nitrate, and sulfate in one or more wells exceeded the limits specified in Table 3-5. The upper-bound concentration of total chromium was slightly above the existing Federal and State MCL of 0.05 mg/L (U.S. EPA, 1988a; DHS, 1989a). Several wells (e.g., MW-114, MW-205, MW-409, MW-486, MW-487, and 11Q2) produced water containing more than 0.05 mg/L of total chromium at least once, with the maximum concentration detected to date equal to 0.1 mg/L (MW-487). These wells are not grouped together at a single location, and we have not yet identified a point source for the elevated concentrations. However, because of variability among subsequent analyses, we are continuing these investigations.

Figure 3-3 is a log probability plot of the median concentrations of total chromium in 164 monitor wells. Analyses of water samples for the two soluble ionic species that make up total dissolved chromium, trivalent chromium (Cr^{+3}), and hexavalent chromium (Cr^{+6}), showed that Cr^{+6} was the predominant species in samples from two wells with elevated total chromium (offsite wells 11Q2 and 11A1). Dresen *et al.* (1987) noted that Cr^{+6} had been used as a corrosion

Table 3-5. Comparisons of upper-bound concentrations of the inorganic and organic constituents detected 10% or more of the time in well waters with the concentrations corresponding to Federal and State maximum contaminant levels (MCLs) and State Action Levels (ALs) for drinking water. Upper-bound concentrations correspond to the 99th cumulative percentile concentration on a lognormal distribution* (see Table 3-4 for lognormal parameters).

Constituent	Units	Regulatory limits for drinking water			Maximum observed concentration from monitored wells[a]	Calculated upper-bound concentration	Is upper-bound concentration greater than Federal or State standard?
		Federal	State				
		MCL	MCL	AL			
Arsenic	mg/L	0.05[b]	0.05[c]		2.7E–02	0.0053	no
Barium	mg/L	1[b]	1[c]		1.2E+00	0.87	no
Benzene	µg/L	5[d]	1[e]		4.5E+04	200	yes
Carbon tetrachloride	µg/L	5[d]	0.5[e]		7.9E+01	13	yes
Chloroform	µg/L	100[f]	100[g]		8.7E+02	51	no
Chromium	mg/L	0.05[b]	0.05[c]		1.0E–01	0.059	yes
cis-1,2-dichloroethylene	µg/L			6[h]	5.0E+01	82	yes
trans-1,2-dichloroethylene	mg/L			10[h]	5.0E+01	11	yes
1,1-dichloroethane	µg/L			5[h]	5.0E+02	16	yes
1,1-dichloroethylene	µg/L	7[d]	6[e]		6.4E+02	57	yes
Copper	mg/L	1[i]	1[j]		4.6E–02	0.023	no
Ethylene dibromide	µg/L		0.02[e]		5.8E+02	1,400	yes
Freon 113	µg/L			1,200[h]	2.9E+02	14	no
Gross alpha	pCi/L	15[k]	15[l]		1.6E+01	11	no
Gross beta	pCi/L		50[m]		2.9E+01	43	no
Iron	mg/L	0.3[i]	0.3[j]		6.7E+00	0.18	no
Lead	mg/L	0.05[b]	0.05[c]		2.7E–01	0.01	no
Manganese	mg/L	0.05[i]	0.05[j]		1.1E+01	0.18	yes
Mercury	mg/L	0.002[b]	0.002[c]		9.0E–04	0.00017	no
Nitrate	mg/L	10[b] (as N)	45[c] (as NO_3)		7.5E+01	370	yes

Table 3-5. (Continued)

Constituent	Units	Regulatory limits for drinking water: Federal MCL	Regulatory limits for drinking water: State MCL	Regulatory limits for drinking water: State AL	Maximum observed concentration from monitored wells[a]	Calculated upper-bound concentration	Is upper-bound concentration greater than Federal or State standard?
Selenium	mg/L	0.01[b]	0.01[c]		1.4E–02	0.0031	no
Sulfate	mg/L	250[i]	250[j]		1.1E+03	280	yes
Tetrachloroethylene (PCE)	µg/L		5[e]		1.8E+03	110	yes
Toluene	µg/L			100[h]	5.6E+04	56	no
Xylene isomers (total)	µg/L		1,750[e]		2.4E+04	4,400	yes
1,1,1-trichloroethane	µg/L	200[d]	200[e]		6.7E+01	8.7	no
Trichloroethylene (TCE)	µg/L	5[d]	5[e]		5.8E+03	1,000	yes
Zinc	mg/L	5[i]	5[j]		2.6E–01	0.051	no

[a]From Appendix Table M-1 of the RI report (Thorpe *et al.*, 1990).

[b]U.S. EPA (1988a).

[c]DHS (1989a).

[d]U.S. EPA (1988b).

[e]DHS (1989b).

[f]Total trihalomethanes, U.S. EPA (1988c).

[g]Total trihalomethanes requirements for community water systems shall comply with the Federal MCL, DHS (1983).

[h]DHS (1989c).

[i]U.S. EPA (1988d).

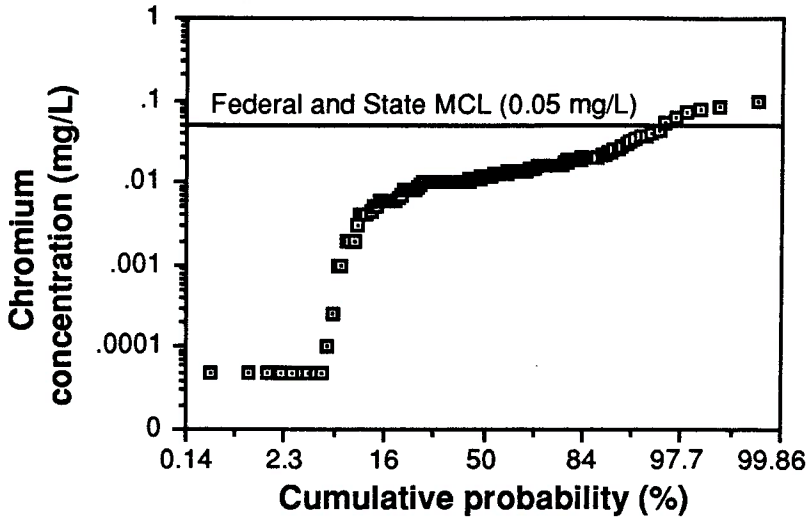
[j]DHS (1989d).

[k]U.S. EPA (1988e).

[l]DHS (1989e).

[m]DHS (1988a).

*99th cumulative percentile (upper-bound) concentration = $GM \times GSD^{2.3}$.



ERD-LSP-91-0009

Figure 3-3. Log probability plot of the median concentrations of total chromium in 164 monitor wells. Fewer than 10% of the wells contain chromium above the Federal and State MCL of 0.05 mg/L.

inhibitor in cooling-tower water at the LLNL site. This water was periodically discharged as blowdown water to storm drains onsite until 1975.

The blowdown discharges may be partially responsible for the elevated concentrations of Cr^{+6}. However, there is insufficient evidence (concentrations of Cr^{+6} in sediments or clearly defined plumes) to determine whether blowdown discharges or natural sources are the actual causes.

As shown in Figure 3-4, of the 161 wells sampled for manganese, 131 or 81% yielded water with no detectable concentrations of that metal. Sixteen of the 161 wells sampled had water containing manganese above the Federal and State secondary drinking water standard of 0.05 mg/L (U.S. EPA, 1988d; DHS, 1989d). This standard is based on organoleptic considerations (U.S. EPA, 1979). Plumes or source terms could not be associated with the wells yielding elevated concentrations of manganese. However, the observed levels are within limits indicative of the ground water in this area. For example, a U.S. Geological Survey report that examined water quality conditions of surface and ground waters in the Livermore–Amador Valley showed that manganese exceeded the secondary standard in 47 of 96 wells tested (Sorenson *et al.*, 1985).

As shown in Figure 3-5, median nitrate concentrations in 13 samples (from 12 wells) exceeded the California drinking water standard of 45 mg/L for nitrate as NO_3 (DHS, 1989a) and the Federal MCL for nitrate of 10 mg/L as nitrogen (U.S. EPA, 1988a). This result is not surprising because elevated levels of nitrate are characteristic of ground waters in the Livermore Valley. Sorenson *et al.* (1985), for example, identified an area about one mile to the west of the LLNL site where wells produce water with nitrate above the standard. Indeed, one well had water with over 80 mg/L of nitrate (20 mg/L as nitrogen). Likely sources of nitrate include

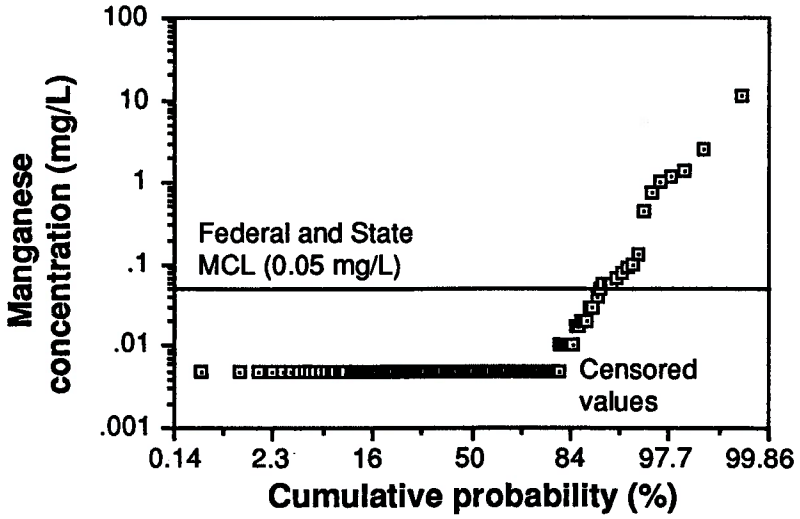


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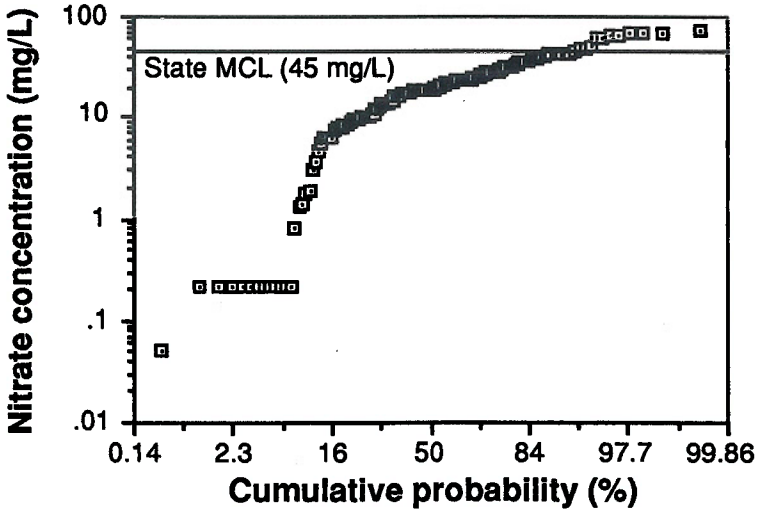
Figure 3-4. Log probability plot of median concentrations of manganese in 161 monitor wells. Most of the wells produced water that did not contain detectable levels of manganese. The Federal and State MCL for manganese is 0.05 mg/L.



ERD-LSP-91-0011

Figure 3-5. Log probability plot of median nitrate concentrations in 161 monitor wells. Nitrate levels exceeded the State MCL of 45 mg/L in 13 samples (from 12 wells). The Federal MCL for nitrate is equivalent to the State MCL, but is expressed as 10 mg/L as nitrogen.

fertilizers leached into soils, organic nitrogen contained in domestic waste waters (septic tanks), and animal wastes. Due to the widespread nature of the nitrate detected in ground waters around the LLNL site (over 90% of the wells had detectable nitrate concentrations), we conclude that the detected nitrate probably arises primarily from agricultural sources.

Sulfate is a ubiquitous constituent of natural waters and was detected in virtually all the wells. Figure 3-6 is a log probability plot of the median sulfate concentrations across all wells. Six of the monitor wells yielded water above the Federal and the State secondary standard of 250 mg/L (U.S. EPA, 1988d; DHS, 1989d). Drinking water with these concentrations of sulfate may have unpleasant organoleptic properties.

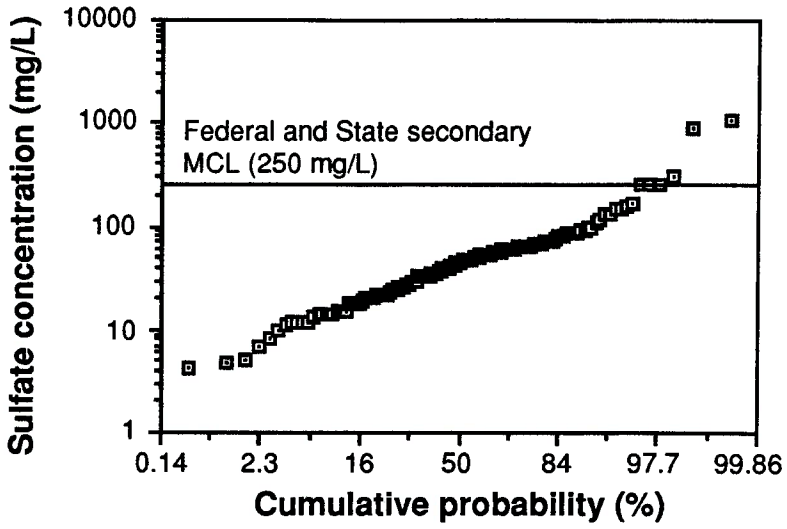
In summary, our analysis of the concentration data for inorganic substances detected in monitor and domestic wells in the vicinity of the LLNL site shows that drinking water standards for some of the substances are exceeded in a few wells. However, except for lead detected in wells completed into the subsurface gasoline spill, and chromium possibly associated with 1960s cooling tower blowdown, none of the other inorganic substances with elevated levels were directly associated with suspected source areas at the LLNL site or had definable ground water plumes. Without definitive data on the occurrence of a plume or sources in the vadose zone, it is not possible to simulate the movement of these substances in ground water.

We suspect that some of the reported high concentrations result from variations in sampling and analysis. Nevertheless, locally elevated concentrations of an inorganic substance are reduced as ground water with lower concentrations of the substance moves through each area, serving to dilute the higher concentrations. Those inorganic substances of concern are noncarcinogens via the oral pathway, based on toxicity data contained in the EPA Integrated Risk Information System (IRIS) (U.S. EPA, 1990a). Thus, dispersion processes should result in concentrations that are below drinking water standards in offsite locations. We will continue to monitor wells with elevated concentrations of inorganic substances, such as chromium, to ensure that the current trends do not change.

3.2.3.2. Organic Constituents

Our review of the concentration data for organic compounds dissolved in ground waters at the LLNL site shows that the various compounds were associated with previous releases of solvents at the site or with the gasoline spill near the former Building 403.

The nongasoline-related organic constituents of ground waters are dominated by halogenated hydrocarbons. The most frequently detected substances in that class were TCE, chloroform, and PCE. Based on our analyses of the concentrations of the VOCs distributed in the saturated zone, we estimate that roughly 64% of the VOC mass in ground water is TCE, 21% is PCE, and 6% is chloroform. The remaining 9% is divided among several different VOCs. Dominant among these other VOCs is 1,1-DCE. Less frequently detected VOCs include 1,1,1-TCA, 1,1-DCA, 1,2-DCA, carbon tetrachloride, and 1,2-DCE (total and cis- and trans-isomers). There is no evidence that any of the organic compounds detected in between 5% and 10% of the samples are known human carcinogens. A complete list of the estimated VOC masses and volumes in ground water is given in the summary table (Table 3-13) at the end of Section 3.2.5.



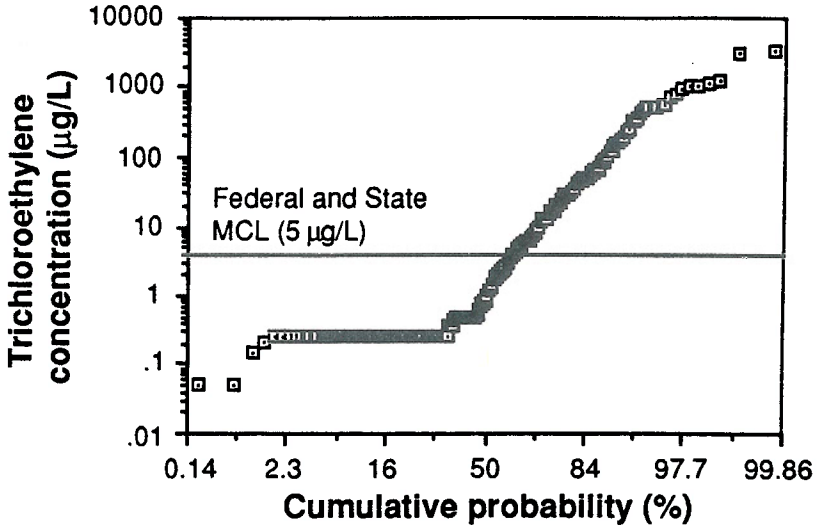
ERD-LSP-91-0012

Figure 3-6. Log probability plot of median sulfate concentrations at monitor wells at the LLNL Livermore site. Six wells exceeded the Federal and State secondary MCL of 250 mg/L.

3.2.3.2.1. Nongasoline-Related VOCs. We detected TCE and chloroform in over 60% of the monitor wells completed at the site. Figures 3-7 and 3-8 show log probability plots of the median concentrations of the time-series concentration data available on these VOCs in each monitor well. About 40% of the wells produced water containing TCE above the Federal and State MCL of 5 μg/L (U.S. EPA, 1988b; DHS, 1989b). For chloroform, less than 10% of the wells exceeded the Federal and State drinking water standard of 100 μg/L for total trihalomethanes (including chloroform, dibromochloromethane, and bromoform) (U.S. EPA, 1988c; DHS, 1983). However, many of the reported concentrations of chloroform appear to be related to low concentrations present immediately after well installation. Such concentrations are probably attributable to the presence of chloroform and other trihalomethanes in municipal water supplies used to mix drilling fluids and for well development. Concentrations of 82 to 381 ppb total trihalomethanes have been detected in LLNL area tap water, with a maximum chloroform concentration of 170 ppb. These results represent five water samples taken from fire hydrants in 1986 and 1987 and analyzed by Brown and Caldwell Laboratories. Additional tap water samples are currently being analyzed.

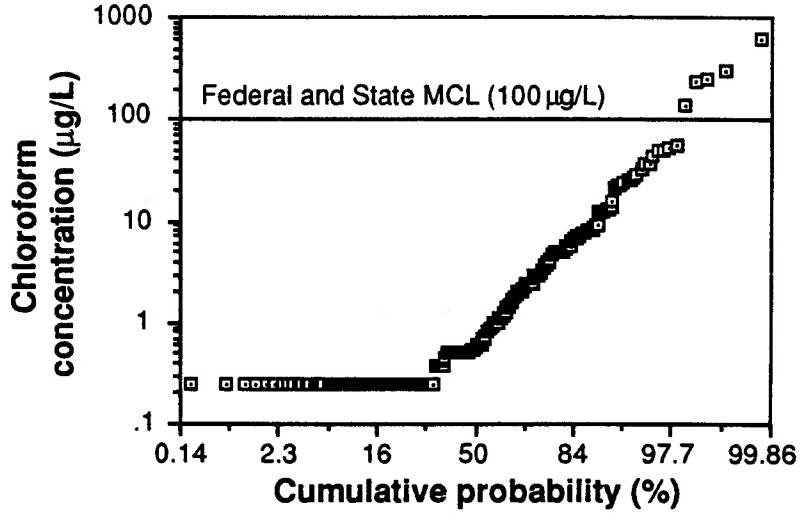
As shown in Figure 3-9, approximately 20% of the median PCE concentrations for the monitor wells exceeded the recently adopted State MCL for PCE of 5 μg/L (DHS, 1989b). The log probability plot for 1,1-DCE in Figure 3-10 shows that about 20% of the median concentrations from the monitor wells exceeded the Federal MCL of 7 μg/L (U.S. EPA, 1988b). Figure 3-11 shows that the Federal MCL of 5 μg/L for carbon tetrachloride (U.S. EPA, 1988b) was exceeded by more than 15% of the median concentrations (from 23 wells), while an additional 40 wells yielded median concentrations that exceeded the State MCL of 0.5 μg/L (DHS, 1989b).

Based on the dominance of TCE, PCE, and chloroform in the ground water (91% of total mass) in the study area, our primary goal for this aspect of our investigation was to assess the



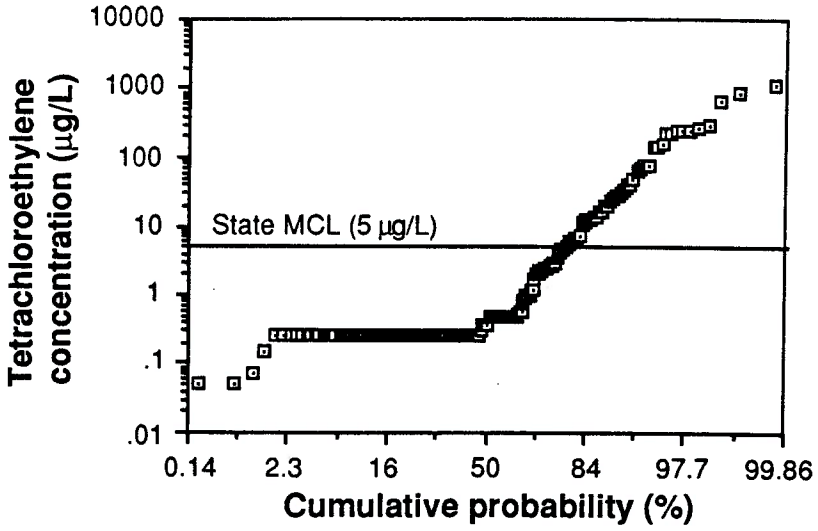
ERD-LSP-91-0013

Figure 3-7. Log probability plot of median TCE concentrations in 267 monitor wells. The Federal and State MCL for TCE is 5 µg/L.



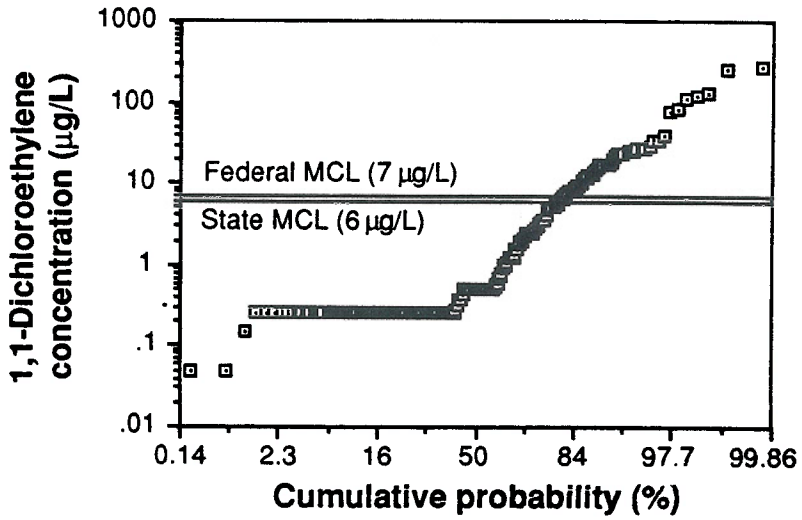
ERD-LSP-91-0097

Figure 3-8. Log probability plot of median concentrations of chloroform across 266 monitor wells. The Federal and State drinking water standards for trihalomethanes, including chloroform, is 100 µg/L.



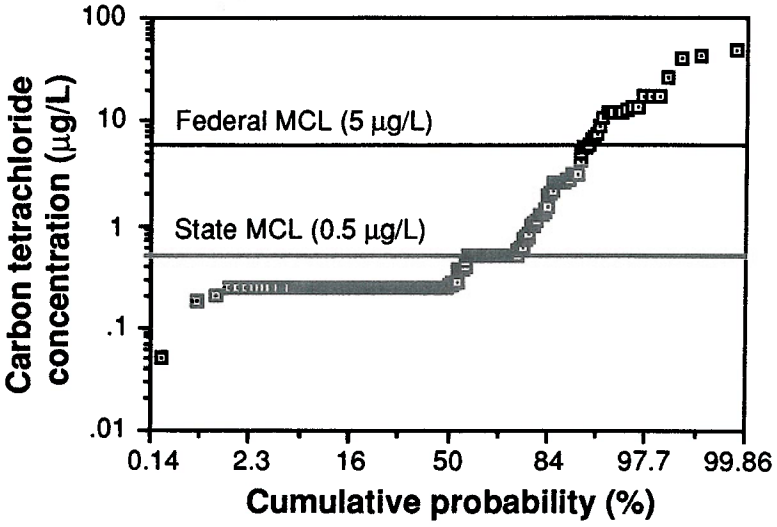
ERD-LSP-91-0098

Figure 3-9. Log probability plot of median tetrachloroethylene (PCE) concentrations across 267 monitor wells. The State MCL for PCE is 5 µg/L. No Federal MCL has been adopted at this time.



ERD-LSP-91-0099

Figure 3-10. Log probability plot of median concentrations of 1,1-DCE across 266 monitor wells. The Federal MCL for 1,1-DCE is 7 µg/L and the State MCL is 6 µg/L.



ERD-LSP-91-0100

Figure 3-11. Log probability plot of median concentrations of carbon tetrachloride across 261 monitor wells. The Federal MCL for carbon tetrachloride is 5 mg/L, whereas the State MCL is 0.5 mg/L.

potential health risks associated with the transport of these substances away from the LLNL site. Each of these VOCs has caused cancer in laboratory animals. All have been considered probable human carcinogens, based on the weight of available evidence. Section 5.1.2 reviews pertinent data on the carcinogenic potential of each of these compounds.

The remaining 9% of the VOC mass (excluding the gasoline spill) is distributed among several different VOCs. Table 3-4 shows the more frequently detected substances in this group. They are carbon tetrachloride (1% of the total VOC mass, with 71 wells having detectable concentrations), 1,1-DCA (1%, 93 wells), 1,1-DCE (5%, 124 wells), total-1,2-DCE (0.5%, 63 wells), and 1,1,1-TCA (0.5%, 90 wells). Table 3-2 shows the organic substances detected at least once, but less than 10% of the time in well-water samples. Among these substances, 1,2-DCA (1% of total VOC mass) was detected at least once in multiple samples in 23% of the wells sampled, and methylene chloride was detected at least once in about 20% of the wells. For both 1,2-DCA and methylene chloride, multiple analyses in a single well frequently yielded no concentrations above detection limits. Thus, the overall frequency of detection was lower than that for the other VOCs.

One method of assessing the potential health risk of this group of substances is to use indicator chemicals that have physicochemical and toxicological properties that are representative of the other VOCs. This general approach is discussed in the *Superfund Public Health Evaluation Manual* (U.S. EPA, 1986a).* To determine which VOC would serve as an acceptable indicator compound, we reviewed data on the distribution of the various compounds in ground water, their physicochemical properties, and their toxicity. The principal component of the remaining VOCs in ground water proved to be 1,1-DCE. It accounts for approximately one-half of the mass of this category and is widely distributed among the monitor wells.

* The most recent guidance document from the EPA for preparing a baseline public health assessment (U.S. EPA, 1989a), which replaces the *Superfund Public Health Evaluation Manual* (U.S. EPA, 1986a), was not published until well after the draft version of this BPHA was submitted for review.

Other substances, such as carbon tetrachloride, 1,1-DCA, 1,2-DCE, and 1,1,1-TCA, were not as widely distributed. Thus, it is difficult to define source regions for the purpose of modeling their transport in ground water. The compounds in this remaining 9% of the VOC mass consist of both carcinogens and noncarcinogens. Carcinogens include carbon tetrachloride, 1,2-DCA, and methylene chloride. Noncarcinogens include 1,1,1-TCA, 1,1-DCA, and 1,2-DCE (U.S. EPA, 1990a).

Evidence for the carcinogenicity of 1,1-DCE is equivocal. Although it has been shown to be positive in genotoxicity assays, the results of chronic bioassays with laboratory animals have not provided conclusive evidence of its carcinogenicity. We therefore treated 1,1-DCE as a noncarcinogen, which is consistent with the approach adopted by the California Department of Health Services (DHS, 1988b). To assess the health risks of the other VOCs in this class, we treated the remaining mass (44%) as though it consisted exclusively of carbon tetrachloride. This compound has the highest carcinogenic potency of all the other compounds.

3.2.3.2.2. Gasoline-Related Compounds. The primary organic components in ground water at the site of the gasoline release are benzene, toluene, and xylene isomers. Table 3-5 shows that upper-bound concentrations of benzene and xylenes in monitor wells across the site (including the Gasoline Spill Area) and the *maximum* reported concentration of toluene (56,000 µg/L) in monitor wells completed adjacent to the subsurface spill site exceeded drinking water standards.

Other minor constituents of gasoline-contaminated ground waters, which were detected less than 10% of the time, include phenol, ethylbenzene, and 1,2-DCA. Phenol exceeded the taste and odor threshold of 1 µg/L established as an action level for chlorinated water supply systems by the State of California (DHS, 1989c) in six wells. The highest concentrations of phenol were detected in wells GSW-5, GSW-20, and GSW-403-6. The highest recorded phenol concentration was 41 µg/L (GSW-403-6). The State of California recently adopted an MCL for ethylbenzene in drinking water of 680 µg/L (DHS, 1989b). We found that maximum concentrations of ethylbenzene exceeded this level in three wells at the site of the gasoline spill. Maximum concentrations of ethylbenzene in wells GSW-5, GSW-403-6, and GSW-16 were 1,000, 780, and 5,900 µg/L, respectively. Concentration data are not available for the time period after the commencement of the pilot remediation effort in the area.

Another co-contaminant in gasoline is 1,2-DCA. The Federal MCL for 1,2-DCA is 3.0 µg/L (U.S. EPA, 1988b), whereas the level recently adopted as an MCL by the State of California (DHS, 1989b) is one order of magnitude lower (0.5 µg/L). In the area of the gasoline spill, 18 wells have median concentrations that exceed the State MCL. However, 1,2-DCA also occurs sporadically in other wells across the site. For example, the median concentrations of 1,2-DCA in 14 wells elsewhere onsite exceed the State MCL. (However, many of these wells had both censored and uncensored concentration data.) Most of the concentration data for ethylene dibromide, also a co-contaminant of gasoline, were censored (<LOD). The highest measured concentration was 580 µg/L, substantially lower than the calculated upper-bound value 1,400 µg/L (see Table 3-5). Concentrations of other minor constituents in well waters in other locations in the study area generally met standards.

The most toxic component of the gasoline spill is benzene, which has been classified as a human carcinogen on the basis of epidemiologic evidence (IARC, 1982a). Evidence is insufficient to classify toluene, ethylbenzene, or the xylene isomers as to their potential human

carcinogenicity. Moreover, the daily intake of benzene that corresponds to a lifetime, incremental cancer risk of 10^{-6} (a one-in-a-million chance of developing cancer) is about 0.03 μg/kg-d, calculated from a potency of 0.029 $(mg/kg\text{-}d)^{-1}$ from the IRIS database (U.S. EPA, 1990a). This intake is considerably below the oral reference doses (RfD) or safe daily intakes for humans for the other gasoline contaminants covered in IRIS (0.1, 0.3, and 2 mg/kg-d for ethylbenzene, toluene, and xylene isomers, respectively). All these values were derived from animal toxicity data for noncarcinogenic endpoints using a safety-factor approach.

To determine whether the benzene component of gasoline constitutes a potential public health hazard via the ground water pathway, we evaluated its potential for offsite transport. This analysis, which is presented in Section 4.3.6.4, indicates that benzene undergoes fairly rapid biodegradation in ground water (half-life of one year or less) and that there is slow movement of ground water in the vicinity of the gasoline spill site (less than 26 ft/y). It is therefore improbable that benzene will ever reach detectable concentrations in ground water offsite. Our review of the environmental chemistry of the other gasoline-related contaminants (toluene, xylene isomers, and ethylbenzene) demonstrated that they, too, have short half-lives (<1 y) as a consequence of degradation in the environment. Because of the limited potential for offsite transport, we conclude that the gasoline contaminants do not constitute a public health concern. Nevertheless, the gasoline spill site will continue to be the subject of ongoing remediation efforts.

3.2.3.3. Radioactive Constituents

Our previous monitoring for radioactive constituents in ground water showed tritium at elevated levels in only a few wells. Historic analyses of gross alpha and beta activities in well waters have been below State MCLs (DHS, 1989e and 1988a) (15 and 50 pCi/L for gross alpha and beta activity, respectively). The single exception was well MW-141, which had 16 pCi/L of gross alpha activity in 1985. When reanalyzed in 1988, gross alpha activity in this well was below detection. Gamma-emitting radionuclides (e.g., ^{137}Cs) were not detected in water samples from 38 wells that were specifically monitored for such radionuclides since 1984 (see Thorpe *et al.* 1990, Appendix Table J-1).

To further investigate the nature and extent of tritium contamination in the vicinity of MW-206, which has had the highest recent concentration of tritium, we analyzed for tritium in an additional 26 wells onsite in 1988 (Dresen *et al.*, 1989). Results of that survey showed that tritium in MW-206 declined from 40,000 pCi/L in 1986 to 28,000 pCi/L in 1988. In 1989, tritium in MW-206 declined further to 25,000 pCi/L (from Thorpe *et al.,* 1990, Table 4.2.3-13). Federal and State MCLs for tritium, by comparison, are both 20,000 pCi/L (DHS, 1988a; U.S. EPA, 1988f). Tritium in MW-363, which is about 60 m to the northwest of MW-206, was detected at a concentration of 21,000 pCi/L in 1988 and 18,000 pCi/L in 1989 (from Thorpe *et al.,* 1990, Table 4.2.3-13). The source area for the tritium in these wells was the taxi-strip waste-storage area, where tritium leaked from evaporation ponds (Buerer, 1983).

The rapid decline in tritium in MW-206 probably reflects the combined effects of radioactive decay (tritium half-life is 12.3 y) and dilution from ground water transport. This trend also suggests that the source of the tritium has been depleted over time because of the remediation work previously completed on the site (Buerer, 1983) and the effect of gradual leaching of tritium from the vadose zone. As tritium in the ground water undergoes further transport and

radioactive decay, concentrations should continue to decline. By the time the ground water reaches the LLNL-site boundary, concentrations will be within background range. Hence, tritium does not constitute a public health hazard. Nevertheless, we continue to monitor these wells for tritium to ensure that the current trend continues.

3.2.4. Persistence and Potential Health Risk of Hazardous Substances in Soils and Sediments

We determined whether any organic, inorganic, or radioactive substances present in surface soils and sediments on the LLNL site and adjacent areas to the west and northwest in nearby arroyos pose a health hazard to either an adult population on the LLNL site or to the public offsite. We evaluated the concentration data obtained from three soil/sediment sampling studies conducted on and near the LLNL site. In this section, we describe the three soil/sediment sampling studies first. Next, for each of the three categories of substances, we present the methodologies we used to assess potential hazards of the various substances. The methodology used to screen contaminants in soils and sediment differs in an important way from that used for screening contaminants in water. Standards do not exist for contaminants in soils or sediment, except for purposes of disposal; thus, concentrations could not be compared to regulatory limits for soil.

For screening purposes, we evaluated the potential health risks from exposure to contaminants in soil by a population onsite, as well as by the public offsite. However, the population onsite is considered to be composed of adults exclusively, and to be conservative, the exposure period for this population is assumed to be a continuous 50 y (as opposed to a workforce scenario, which might also be estimated conservatively at 50 y of exposure, but would take into account interruptions by weekends, holidays, vacations, sick leave, and changes in job assignment). The purpose for addressing "adult onsite" (aos) exposure is to account for individuals who through the course of their employment at LLNL might come into contact with contaminated soils, albeit periodically. Sediment below the surface layer has no potential for direct exposure and is considered only as a potential pathway for contaminants to reach the ground water.

The reasons for addressing adult onsite exposure exclusively are because:

1. Ownership of the LLNL property is not likely to transfer from the DOE in the foreseeable future.
2. Considering the sizeable number of structures on the Livermore site, it is not reasonable to assume that the property would ever become residential, even if the DOE did transfer ownership.
3. Regardless of any current or possible future use of the LLNL site, the DOE is committed to ensuring, in perpetuity, that the LLNL property not pose a public, occupational, or environmental hazard.

Furthermore, any change of ownership of the Livermore site or any portion thereof shall be consummated by the DOE only if there is provision for continued maintenance of any containment system, treatment system, monitoring system, or other response action(s) installed or implemented pursuant to the Federal Facility Agreement Under CERCLA/SARA Section 120 (U.S. EPA *et al.*, 1988).

3.2.4.1. Soil/Sediment Sampling Studies

The first soil-sampling study was conducted in 1984. This study was a special assessment to determine the possible source(s) of chlorinated hydrocarbons discovered in 1983 in 8 of 20 ground water wells. Three of the eight wells were drinking water wells on properties adjacent to LLNL. As part of this special assessment, which is described by Carpenter (1984), a total of 108 soil/sediment sampling holes were drilled on or adjacent to the LLNL Livermore site. Because potential exposures arise from concentrations on surface soils from the LLNL site, only the concentration data reported for near-surface soil samples from boreholes drilled on the LLNL site or on immediately adjacent offsite properties (now part of the LLNL buffer zone) were used. Such data are provided by Carpenter (1984) for acetone, methylene chloride (dichloromethane), trichlorotrifluoroethane (Freon 113), methyl ethyl ketone, 1,1,1-TCA, TCE, PCE, and xylene isomers. These data are from 78 different locations on the LLNL site, including the former Righetti property, which is now part of LLNL's western buffer zone. All these near-surface soil samples were obtained from depths that ranged from about 0.8 to 1.2 m. In Figure 3-12, the solid circles and corresponding location identification numbers show the general locations where these soil sampling holes were drilled. A weighed aliquot of a soil sample was analyzed using EPA Methods 601, 602, and, in some cases, 624. This was done because at the time this sampling was performed, soil methods were not yet prescribed by the EPA. These methods are briefly described and compared in Appendix B of the report by Carpenter (1984). That appendix also gives the complete list of organic chemicals analyzed by each method. These analyses are similar to the methods now recommended by the U.S. EPA for soils described in the publication *Test Methods for Evaluating Solid Wastes* (U.S. EPA, 1986b).

The second soil/sediment sampling effort was conducted as part of the annual environmental monitoring program for the LLNL site for 1988 and 1989. This sampling was performed in 1988 and 1989 at five locations at LLNL and at one location on the east side of the Sandia National Laboratories (SNL) Livermore property, located to the south of LLNL. The five sediment sampling locations associated with the LLNL site that are of interest are indicated by solid triangles and labeled in Figure 3-12 as:

- Arroyo Las Positas East (ALPE).
- Arroyo Las Positas West (ALPW).
- Arroyo Seco North (ASN).
- Arroyo Seco South (ASS).
- Central Drainage Basin (CDB).

Sediment sampling location Arroyo Seco East (ASE) is on SNL property and is not shown in Figure 3-12.

As part of the annual environmental monitoring program, storm-water runoff was also sampled at locations near the sites of sediment sampling. These storm-water runoff sampling locations are included in Figure 3-12 and are indicated by open boxes that are labeled numerically as 1 through 3, 5, and 6. Location 4 is at the site of sediment sampling located on SNL, Livermore property, and is not shown. The soils were analyzed for organic chemicals using EPA Methods 8010, 8020, and 8080 (U.S. EPA, 1986b). The Total Threshold Limit Concentration (TTLC) for inorganic substances, described in the California Code of Regulations

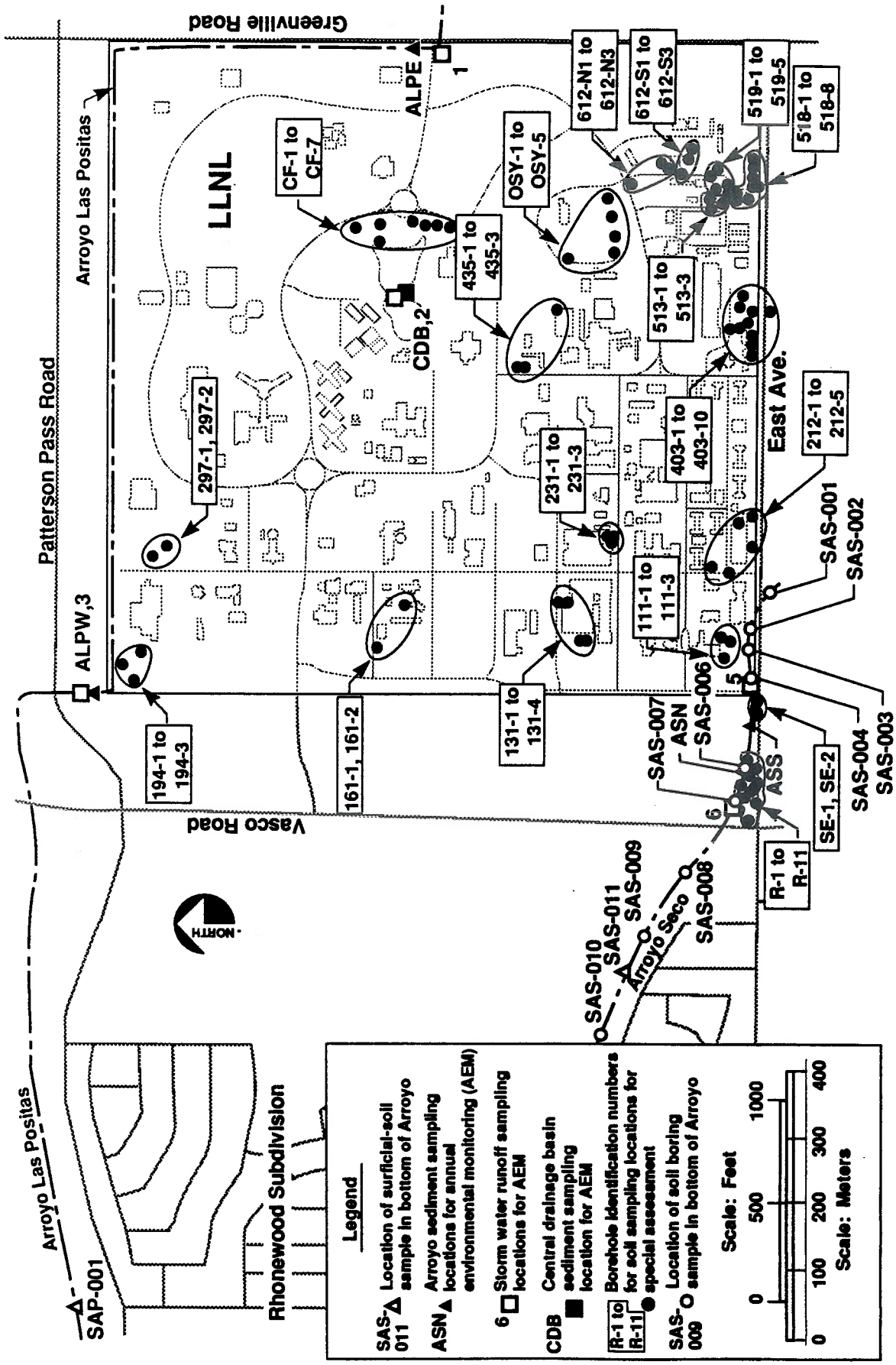


Figure 3-12. Sample location points at LLNL and adjacent offsite area.

(DHS, 1984), and the concentration of selected radioactive substances were determined for the soils from the five sampling locations on the LLNL site. The analytical chemistry for all of the organic and inorganic chemicals was performed for LLNL by Brown and Caldwell Laboratories (Emeryville, California). These data are presented in Appendix N of the RI report (Thorpe *et al.*, 1990) and are contained in an electronic data base that is under the administrative control of the LLNL Environmental Protection Department (LLNL, 1990). However, the concentration data for selected radioactive substances in the soils onsite and offsite that were measured as part of the annual environmental monitoring effort for 1988 are currently only reported by Brekke *et al.* (1989) but also appear separately in Appendix Tables N-6 and N-7 of the RI report (Thorpe *et al.*, 1990).

A third soil/sediment sampling study was conducted in 1989 as part of the ongoing LLNL site remedial investigations. Soils to a depth of about 15 cm were sampled at locations onsite and offsite. Offsite soil- and sediment-sampling locations were located in the Arroyo Seco and the Arroyo Las Positas close to the Rhonewood residential area. These offsite sampling locations have SAS and SAP identifiers, respectively, in Figure 3-12. The intended sampling at location SAS-005 was not performed due to mechanical difficulties. The remaining sampling locations were along the storm-drain system (SSDs) and from surficial soils (SSSs) at the LLNL site (see Fig. 3-13). All the soils were analyzed for organic chemicals using EPA Methods 8240, 8270, and 8080 (U.S. EPA, 1986b). The Soluble Threshold Limit Concentration (STLC) and the TTLC for inorganic substances, as described in the California Code of Regulations (DHS, 1984), as well as the concentrations of selected radioactive substances, were determined for the soil samples. As part of the LLNL-site remedial investigations, storm-water runoff was also sampled along the surface drainage system onsite. Appendix N in the RI report (Thorpe *et al.*, 1990) contains the soil/sediment and storm-drain water sampling data from the above studies and includes tables of all chemicals looked for, but not detected.

3.2.4.2. Organic Chemicals

We followed a methodology similar to the one adopted for the screening of substances in ground water. We began our analysis of the concentration data for organic substances in soils by combining the data from the three soil/sediment sampling studies just described. We then determined the frequency with which each of the organic compounds was detected at least once at a concentration above the LOD. The detection frequencies were then separated into two categories similar to the ones used in the screening of substances detected in ground water. The first category included substances detected at less than 10% of the sampling locations. The second category included substances detected at greater than or equal to 10% of the sampling locations. These two categories were selected to distinguish those substances with the least exposure potential (detection frequency <10%) from those substances with the greatest exposure potential (detection frequency $\geq$10%). All detection frequencies were calculated by dividing the number of locations having a detectable concentration of a particular compound by the total number of locations at which a soil/sediment sample was taken. An analysis was then performed specifically for that compound. Because the number of chemical analyses we performed was not the same in all three studies, the total number of sampling locations varies for compounds.

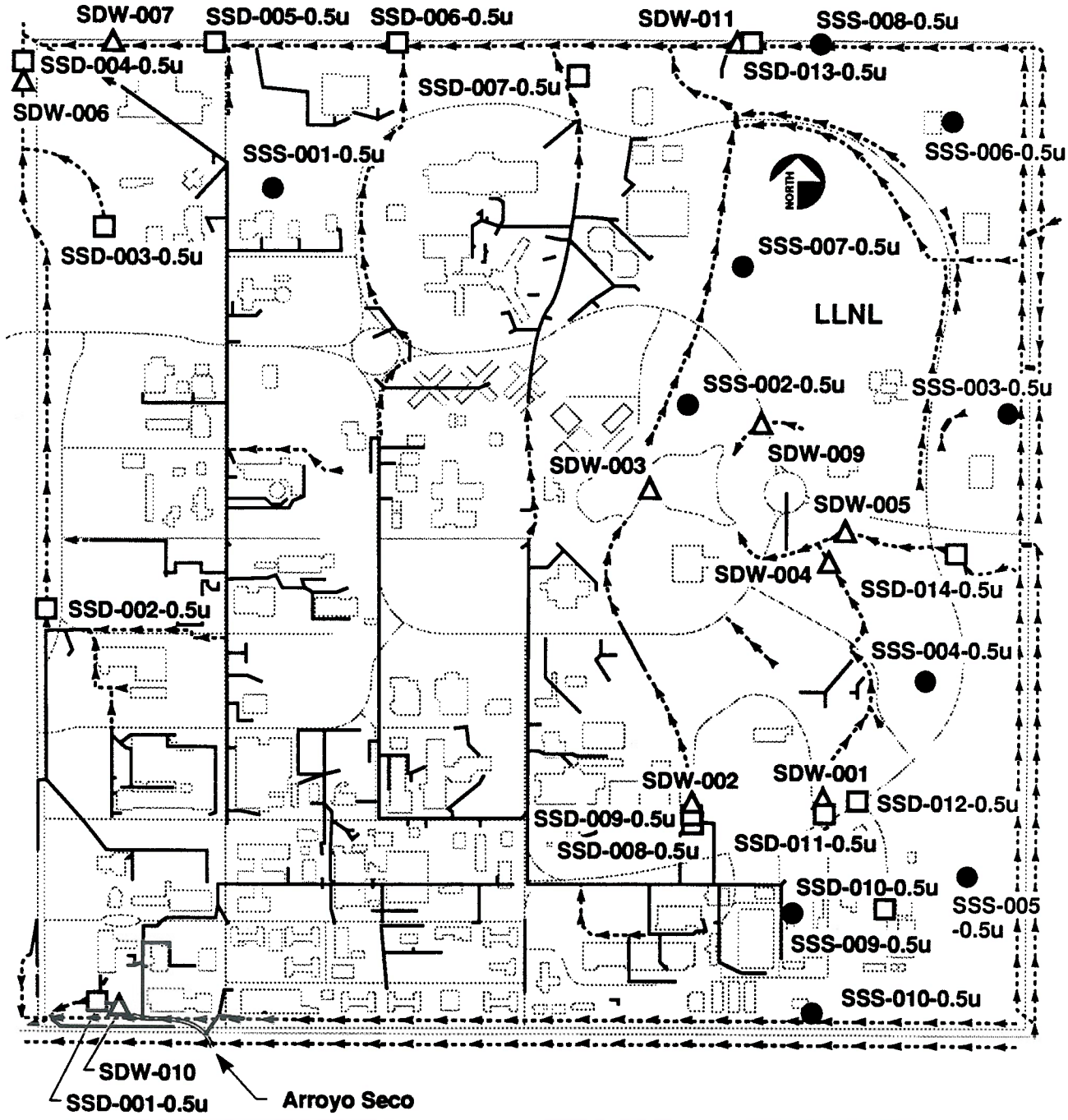


Figure 3-13. Surface soil and surface water sampling locations at the LLNL Livermore site (SDW-008 was a trip blank).

Table 3-6 lists the organic chemicals detected at less than 10% of the locations. The number of times each substance was detected, the number of soil/sediment sampling locations for each substance, and the actual percentage of locations at which the substance was detected are also shown. The detection frequencies for these organic substances range from as low as 2% of 61 sampling locations for 1,1-dichloroethane and 2% of 47 sampling locations for lindane to as high as 9% of 118 sampling locations for 1,1,1-trichloroethane.

Table 3-7 lists the chemicals detected at greater than or equal to 10% of the sampling locations. This table also shows the number of times each substance was detected, the number of soil/sediment sampling locations for each substance, and the actual percentage of locations at which the substance was detected. The highest detection frequency for these organic substances was 100% of one sampling location for methyl acetate and 34% of 118 sampling locations for PCE. The remainder of the organic chemicals in Table 3-7 had detection frequencies ranging from as low as 10% of 29 sampling locations to 32% of 118 sampling locations.

Only fluoranthene and pyrene, which are naturally occurring organic chemicals found in soil (Dragun, 1988), were detected offsite and only in one soil/sediment sample taken from the Arroyo Seco west of the LLNL site (SAS-011 in Fig. 3-12). The concentrations offsite of both fluoranthene and pyrene that were detected were at levels just above the LOD [0.1 mg/kg; see Appendix N of the RI report (Thorpe *et al.*, 1990)]. These compounds were detected at two and three sampling locations on the LLNL site, respectively, but are not likely to be found in ground water because they are strongly sorbed to organic material in soils (Dragun, 1988), effectively limiting their mobility through the soil. We detected no other organic chemicals at the two surficial soil/sediment sampling locations in the arroyos to the west (Arroyo Seco) and northwest (Arroyo Las Positas) of the site. Consequently, it is not likely that individuals living in the Rhonewood residential subdivision will be exposed to soil contaminated with organic chemicals that originated at the LLNL site.

Our finding that PCE and TCE were the most prevalent substances (i.e., in terms of frequency of detection and number of sampling locations) in the soils at the LLNL site is consistent with the results of our screening analysis of substances in ground water. On the basis of local environmental and geophysical conditions, both PCE and TCE are relatively mobile in soils and sediments and, therefore, could reach ground water if there is a liquid flux through the unsaturated zone. Furthermore, the presence of detectable levels of benzene, toluene, and total xylene isomers at over 10% of the sampling locations (Table 3-7) is also not surprising for two reasons. First, these organic substances are gasoline-related compounds typically found in soils that come into contact with street runoff. Second, paved surfaces have come to dominate the LLNL landscape (excluding the recently acquired buffer zone), and most of the pavement is used as roads or for parking motor vehicles.

The frequency-of-detection data presented above indicate that there is only a small likelihood of exposure to soils contaminated with organic chemicals for adults onsite (aos) and virtually no possibility of exposure to populations offsite. Furthermore, it is unlikely that storm-water runoff from the LLNL site would introduce significant concentrations of organic chemicals onto surface soil offsite. Moreover, few organic chemicals have been detected in water samples from storm drains on the LLNL site [see SDW samples in Appendix Table N-9 of the RI report (Thorpe *et al.*, 1990)]. Additionally, with only one exception (acetone), the concentrations of the detected

Table 3-6. Organic chemicals detected at less than 10% of the soil/sediment sampling locations at the LLNL site or on adjacent properties.

Chemical	Number of soil/sediment samples with detectable concentrations	Number of soil/sediment sampling locations for compound	Percent detected[a]
Benzo(a)anthracene	1	29	3.4
Benzo(a)pyrene	1	29	3.4
Benzo(b)fluoranthene	1	29	3.4
Benzo(g,h,k)perylene	1	29	3.4
Benzo(k)fluoranthene	1	29	3.4
Bis(2-ethylhexyl)phthalate	1	29	3.4
Carbon disulfide	1	28	3.6
Chrysene	1	29	3.4
1,3-dichlorobenzene	2	59	3.4
1,1-dichloroethane	1	61	1.6
Dimethylphthalate	1	29	3.4
Endosulfan I	1	47	2.1
Fluoranthene	2	29	6.9
Lindane	1	47	2.1
Phenanthrene	2	29	6.9
1,1,1-trichloroethane	11	118	9.3

[a]Number of samples with detectable concentrations divided by the number of sampling locations for the compound multiplied by 100.

Table 3-7. Organic chemicals detected at greater than or equal to 10% of the soil/sediment sampling locations at the LLNL site or on adjacent properties.

Chemical	Number of soil/sediment samples with detectable concentrations	Number of soil/sediment sampling locations for compound	Percent detected[a]
Acetone	9	88	10.2
Aroclor (PCB) 1254	10	47	21.3
Benzene	13	62	21.0
Chloroform	19	61	31.1
1,2-dichlorobenzene (total)	15	59	25.4
1,2-dichloroethylene	7	61	11.5
Ethylbenzene	19	61	31.1
Methyl acetate	1	1	100.0

Table 3-7. (Continued)

Chemical	Number of soil/sediment samples with detectable concentrations	Number of soil/sediment sampling locations for compound	Percent detected[a]
Methyl ethyl ketone	10	88	11.4
Methylene chloride (dichloromethane)	21	118	17.8
Pyrene	3	29	10.3
Tetrachloroethylene (PCE)	40	118	33.9
Toluene	20	62	32.3
Trichlorofluoromethane (Freon 11)	17	61	27.9
Trichloroethylene (TCE)	38	118	32.2
Trichlorotrifluoroethane (Freon 113)	14	88	15.9
Xylene isomers (total)	23	116	19.8

[a]Number of samples with detectable concentrations divided by the number of sampling locations for the compound multiplied by 100.

organic chemicals in the storm drains were low (below 10 μg/L). Finally, the measurable concentrations of these substances were found at only one or two (e.g., acetone) storm-drain water sampling locations. Therefore, a source that might sustain these concentrations over time is unlikely.

Because regulatory limits for organic chemicals in soils have not been established, we adopted another evaluation procedure. This procedure is designed to ensure that none of the organic substances at concentrations above the LOD represents a significant, potential health concern for adults onsite or for the public offsite. The evaluation procedure we selected first involves the development of unit pathway-exposure factors (PEFs) for contaminated soils. For a given exposure pathway, a PEF translates the concentration of an organic chemical in soil or air into an equivalent lifetime soil-based exposure (in units of mg/kg-d) for the individuals of a population over a given period of time. This period is either the lifetime for the public or a time-weighted, uninterrupted duration of employment for LLNL adults onsite. We then use the following equation to calculate a maximum value for each equivalent, lifetime soil-based exposure, $e_{i\text{-max}}$, applicable to a population at risk:

$$e_{i\text{-max}}(\text{population at risk}) = C_{\max} \times F_i , \qquad (3\text{-}1)$$

where F_i is a pathway-exposure factor for pathway i, and $C_{\max}$ is the maximum recorded concentration of a contaminant in soil or air.

There are four pathways for which $C_{\max}$ must be determined:

- Ingestion of soil particles.
- Dermal absorption.

- Inhalation of soil particles.
- Inhalation of chemicals volatilized from soil to the atmosphere.

For ingestion of soil particles ($i = 1$), as well as for dermal absorption of organic chemicals from soil particles deposited on exposed skin surfaces ($i = 3$), C_{max} in Eq. (3-1) is equal to the maximum concentration of an organic chemical reported in soil, denoted $C_{s\text{-}max}$. For inhalation outdoors of soil particles ($i = 2a$), C_{max} in Eq. (3-1) is equal to the maximum concentration of an organic chemical on soil particles in air, denoted $C_{p\text{-}max}$. For inhalation of chemicals volatilized to the atmosphere from soil ($i = 2b$), C_{max} in Eq. (3-1) is equal to the maximum concentration of an organic chemical detected in air that is attributed to exhalation from soil, $C_{a\text{-}max}$. The $C_{s\text{-}max}$ values for the organic chemicals at concentrations above the LOD in soil are obtained directly from the soil/sediment sampling studies. However, both the $C_{p\text{-}max}$ and the $C_{a\text{-}max}$ values for these chemicals must be estimated. The derivation of these estimates for the organic chemicals of concern is described in Appendix A.

To determine the value of a PEF (F_i) for use in Eq. (3-1), we made several conservative assumptions for screening purposes. First, we assumed that the exposure period for the public for any exposure pathway of concern is equal to a 70-y lifetime. As explained earlier, we assumed that the exposure period for adults onsite for any pathway of concern is equal to a continuous, 50-y duration of employment and that adults onsite have 70-y lifespans. Furthermore, a PEF for the public for a 70-y lifetime, F_i(public), is based on the corresponding component PEFs for children, f_i(child), and adults, f_i(adult). A PEF for adult-onsite exposure, F_i (aos), is computed only on the basis of the corresponding component PEF for an adult, f_i(adult). For example,

$$F_i(\text{public}) = \left[\frac{15}{70} \times f_i(\text{child})\right] + \left[\frac{55}{70} \times f_i(\text{adult})\right], \tag{3-2}$$

and

$$F_i(\text{aos}) = \frac{50}{70} \times f_i(\text{adult}) . \tag{3-3}$$

In Eq. (3-2), the factors 15/70 and 55/70 are estimates of the fraction of a 70-y lifespan an individual spends as a child and an adult, respectively. In Eq. (3-3), the factor 50/70 represents the fraction of a 70-y lifespan an adult at the LLNL site works outdoors. Because public access to the LLNL site is controlled, the primary potential pathway by which the public offsite may be exposed to organic substances in soils is via the inhalation of VOCs that volatilize from sources onsite and are subsequently transported to adjacent areas downwind. For the purposes of our screening analysis, we assessed the potential exposure to TCE exhaled from soils adjacent to Building 518, the area with the highest known concentrations of VOCs in soil. Only the concentrations of TCE in the surface soil near Building 518 were considered because TCE is by far the dominant VOC at this location. Unless there is a continuing source, actual concentrations would diminish after a few years due to volatilization.

For LLNL adults onsite, we developed PEFs for ingestion of soil particles, F_1(aos), for inhalation outdoors of soil particles, F_{2a}(aos), and for dermal absorption of chemicals from soil particles deposited on exposed skin surfaces F_3(aos). We also calculated a PEF term addressing

inhalation of TCE exhaled from the soil adjacent to Building 518 for adults onsite outdoors in that area, F_{2b}(aos). Each PEF of interest was developed according to the methods described by McKone (1988) for estimating multipathway exposure to environmental contaminants. These procedures are explained in more detail in Appendix A.

Our next step in the evaluation procedure was to add the relevant $e_{i\text{-max}}$ values for each organic chemical and population. Accordingly, the $e_{i\text{-max}}$ value for the relevant exposure pathway applicable to the public is equal to a total equivalent maximum lifetime exposure to the maximum concentration of an organic chemical in soil, E^{org}_{max}(public). Similarly, summation of the $e_{i\text{-max}}$ values for the relevant exposure pathways applicable to an adult onsite outdoors continuously for 50 y yields a total equivalent maximum exposure to the maximum concentration reported for an organic chemical in soil, E^{org}_{max}(aos).

The final step of this screening process is to evaluate the maximum soil-based total daily dose of organic chemicals from all relevant exposure pathways, $E^{org}_{s\text{-max}}$, for adults onsite and for the public, in terms of noncarcinogenic hazard and carcinogenic risk. The procedure for accomplishing this evaluation is described by the EPA in the Superfund Public Health Evaluation Manual (U.S. EPA, 1986a). In summary, the noncarcinogenic hazard is determined by dividing the chemical-specific E^{org}_{max} value for adults onsite or the public offsite by the chemical-specific reference dose (RfD), which is considered to be a conservative estimate of the acceptable daily intake for that chemical.

The result of this division is a measure called the hazard index. Any single chemical with an exposure level greater than the reference dose level will yield a hazard index greater than one. Should this occur, there may be concern for a potential health risk. Furthermore, for multiple chemical exposures, the hazard index can still exceed one, even if no single chemical exceeds its acceptable level. This too would indicate a concern that a potential health risk might exist, although the assumption of additivity is most properly applicable to compounds that induce the same health effects by identical mechanisms (U.S. EPA, 1986a).

Carcinogenic risk is determined by multiplying the chemical-specific E^{org}_{max} value for adults onsite or the public offsite by the chemical-specific maximum cancer potency factor (CPF_{max}), which is defined as the "slope factor," or unit risk per dose rate, which results from application of a low-dose extrapolation procedure to laboratory animal data. According to the revised National Oil and Hazardous Substances Contingency Plan promulgated by the U.S. EPA (1990b), the sum of the cancer risks for all individual chemicals should not exceed a target risk level between 10^{-4} and 10^{-6}. Additive risk that exceeds this level suggests a potential health problem for average individuals in exposed populations.

We have applied a procedure similar to the EPA–recommended evaluation procedure to the organic chemicals detected above the LOD on the LLNL Livermore site, with a frequency that, for purposes of conservatism, is greater than or equal to 5% (rather than 10%; see Tables 3-6 and 3-7). This selection process eliminated from further consideration only those organic chemicals that were detected just once or twice in soil samples from the LLNL Livermore site. Because of the very limited occurrence of these compounds, there is no reason to believe they represent any potential threat to the health and safety of adults onsite.

The results of applying our evaluation procedure to the organic substances occurring at a frequency greater than or equal to 5% appear in Table 3-8, beginning on page 3-36. The total for

the hazard index for noncarcinogenic effects and the total for the risk related to carcinogenic effects are less than unity (i.e., 2.6×10^{-3}) and between 10^{-4} and 10^{-6} (i.e., 1.9×10^{-5}), respectively. These results indicate that these chemicals individually and collectively in soil do not pose a health hazard to adults onsite. Furthermore, this screening assumes continued exposure to the highest concentration measured, which in each case is extremely localized and not an actual work site. Appendix B contains tables of exposures and associated hazards and risks for organic chemicals computed using the typical EPA methodology and parameters.

According to the screening analyses presented in Table 3-8 (see pp. 3-36 and 3-37), the polychlorinated biphenyl Aroclor 1254 at sample location SSD-008 contributes significantly to both the noncarcinogenic-hazard index and the carcinogenic-risk level for adults onsite. The soils from the locations identified as having the highest concentrations of Aroclor 1254 (SSD-008/SSD-009, which are duplicates from the same location, and SSS-009; see Fig. 3-13) have already been removed and disposed of. We note that eliminating Aroclor 1254 from these sites reduces the screening level maximum noncarcinogenic-hazard index for adults onsite from 2.6×10^{-3} to 8.8×10^{-4} and lowers the screening level maximum carcinogenic-risk for adults onsite from 1.9×10^{-5} to 5.1×10^{-6}. Additional soil sampling will be conducted to determine if any concentrations of Aroclor 1254 detected in soil samples onsite (> LOD) are the result of an old source that has been cleaned up or an existing source that requires remediation. Additionally, administrative controls are in place to prevent adults onsite from working continuously in the area identified as having had elevated levels of Aroclor 1254 in the soil.

For the public offsite, the value for E^{org}_{max}(public) is 7.4×10^{-10} mg/kg-d (see Appendix A). As explained earlier, this is a conservative value for E^{org}_{max} (public) and is based on public exposure to TCE exhaled from contaminated soil near Building 518 and transported offsite. An RfD for TCE is not available, so a hazard index could not be determined. However, the screening risk value for E^{org}_{max}(public) can be calculated and is lower than a lifetime cancer risk of 10^{-6} (i.e., 1.3×10^{-7}). Accordingly, VOCs, such as TCE, in soils on the LLNL site do not constitute a potential health hazard to the public offsite.

3.2.4.3. Inorganic Chemicals

As mentioned previously, there are no Federal or State limits, similar to MCLs, for chemicals in soil that is not being excavated and removed for disposal. However, the State of California (DHS, 1984) does list TTLC values (based on nitric acid digestion) and STLC values (based on citric acid extraction) for substances in waste material, such as excavated soil, drilling muds, or incinerator ash. Accordingly, these analytical methods are commonly used to monitor concentrations of inorganic chemicals in soil for CERCLA investigations, even though such soils generally are not waste material. Thus, our analyses for inorganic substances in soils at locations onsite and offsite involved using both TTLC and STLC extraction methods [see Appendix N of the RI report (Thorpe *et al.*, 1990)].

To determine if the the inorganic chemicals that were detected onsite or offsite pose a potential noncarcinogenic hazard or carcinogenic risk to adults onsite or to the public offsite, the following procedure was followed. We first determined the frequency with which each of the inorganic compounds was detected. The purpose of this exercise was to compare the number of times a particular inorganic chemical was detected (by the TTLC method) in soil with the total number of soil samples in which it was looked for in order to establish generally the geographical

distribution of each substance around the LLNL Livermore site and adjacent properties. Table 3-9 contains the results of this procedure.

According to the data presented in Table 3-9, over half (10/18) of the substances detected (by the TTLC method) above the LOD occurred at all locations from which soil samples were taken [see also data base in Appendix N of the RI report (Thorpe *et al.*, 1990)]. Moreover, of the remaining substances detected above the LOD, beryllium (nearly three-quarters of 57 samples), cadmium (more than half of 57 samples), and mercury (almost half of 57 samples) can be considered to be fairly widely dispersed; whereas, silver (only about one-quarter of 57 samples) and antimony (more than one-sixth of 57 samples) are less widely dispersed, and selenium (only 1 of 49 samples), thallium (only 3 of 49 samples), and molybdenum (only 5 of 57 samples) occur infrequently. However, each of these inorganic substances are naturally occurring and are likely to appear anywhere in trace amounts. Therefore, it remains necessary to determine how the concentrations of each of the substances, even those detected only a small number of times, compare to respective background levels.

To determine if any of these concentrations are consistent with background concentrations, we followed the methodology of Michels (1971) for statistically analyzing sampling data (see Appendix C). This procedure allows distinctions to be made between background distributions and increments from a local source (see Figs. C-1 through C-19 in Appendix C). Applying this analytical method to the sampling data for inorganic chemicals and to data from Site 300 and selected California counties (California Energy Commission, 1985) (see Appendix C for these data), we determined that barium, selenium, thallium, and vanadium were not distinct from background levels. Titanium was not analyzed using this procedure because of insufficient data. Accordingly, Tables 3-10 and 3-11 contain the results of our screening for potential noncarcinogenic hazard and carcinogenic risk to adults onsite from exposure to the remaining 13 inorganic chemicals identified in Table 3-9 and which we consider to be at concentrations that may be distinct from background levels (see Appendix C).

Although the total noncarcinogenic hazard for the inorganic chemicals of concern for adults onsite is greater than unity in Table 3-10, categorizing each chemical according to its target organ and subtotaling the respective hazard indices accordingly yields groupings with hazard indices that do not exceed unity (see Table 3-11). Consequently, we conclude that the noncarcinogenic hazard to adults onsite is not significant on the basis of the data in Table 3-11.

The data in Table 3-10 show that concentrations found in two sample sites where sediment has accumulated, SSD-009 and SSS-009, contribute significantly to the noncarcinogenic hazard potential. As noted earlier, the soils at these two locations have been removed, eliminating those concentrations from the set of current possible exposures. A calculation of the cumulative hazard index using the next highest concentrations found onsite yields 2.8×10^{-1}, less than unity.

The total cancer risk for adults onsite from exposure to inorganic chemicals is presented in Table 3-12, and these data suggest a significant cancer risk might exist to persons directly exposed to these chemicals. This estimate of cancer risk is a conservative one, however, and may be substantially lower for the following reasons.

Table 3-8. Screening analyses for soil-based exposures to organic chemicals by adults onsite

Organic chemicals detected with a frequency ≥ 5% at LLNL site	Location[a]	Maximum soil concentration, $C_{s\text{-}max}^{org}$ (mg/kg)[b]	Maximum total daily dose (all exposure pathways), $E_{s\text{-}max}^{org}$ (mg/kg-d)	Reference dose, RfD (mg/kg-d)
Acetone	SSS-009	8.0E+00	1.5E–05	1.0E–01[d]
Aroclor (PCB) 1254	SSD-008	1.3E+00	2.4E–06	1.0E–03[e]
Benzene	ALPW	1.1E–02	2.0E–08	Pending
Chloroform	ASS	9.5E–03	1.8E–08	1.0E–02[d]
1,2-dichlorobenzene	ASN	8.7E–03	1.6E–08	9.0E–02[d]
1,2-dichloroethylene (total)	ALPE	1.6E–03	3.0E–09	2.0E–02[d,j]
Ethylbenzene	ALPW	5.2E–03	9.6E–09	1.1E–01[d]
Fluoranthene	SSS-009	1.8E+00	3.3E–06	No data
Methyl acetate	SSD-006	2.0E+00	3.7E–06	1.9E+01[d]
Methylene chloride (dichloromethane)	161-2	5.0E–02	9.3E–08	9.0E–01[l]
Methyl ethyl ketone	612-S2, 612-S3	4.0E–01	7.4E–07	9.0E–02[l]
Phenanthrene	SSS-009	1.0E+00	1.9E–06	No data
Pyrene	SSS-009	1.3E+00	2.4E–06	No data
Tetrachloroethylene (PCE)	518-1	3.7E–01	6.8E–07	1.0E–02[d]
Toluene	ALPE	8.3E–03	1.5E–08	6.0E–01[l]
1,1,1-trichloroethane (1,1,1-TCA)	513-1	1.4E–01	2.6E–07	3.0E–01[l]
Trichloroethylene (TCE)	518-2	3.0E+00	7.9E–06[o]	Pending
Trichlorofluoromethane (Freon 11)	SSD-013, SSD-014	3.0E–01	5.6E–07	3.0E–01[d]
Trichlorotrifluorethane (Freon 113)	513-1	1.6E–01	3.0E–07	3.0E+01[d]
Xylene isomers (total)	SSD-006, SSD-010, SSD-013, SSD-014	2.0E–01	3.7E–07	2.0E+00[d]
				Total

for a continuous 50-y period.

Hazard index for noncarcinogenic effects, HI [$E^{org}_{s\text{-}max}$/RfD]	Target	Maximum cancer potency factor, CPF_{max} (1/[mg/kg-d])	Risk related to carcinogenic effects, R [$E^{org}_{s\text{-}max} \cdot CPF_{max}$]	Weight-of-evidence grouping; target[c]	Reference
1.5E–04	Liver, kidney	Under review			U.S. EPA, 1990a
2.4E–03	NOAEL[f]	7.7E+00[g,h]	1.8E–05	B2; liver	Geyer *et al.*, 1986 & U.S. EPA, 1990a
		2.9E–02[h]	5.8E–10	A; blood	U.S. EPA, 1990a
1.8E–06	Liver	8.1E–02[i]	1.5E–09	B2; kidney	U.S. EPA, 1990a
1.8E–07	Kidney	Pending			U.S. EPA, 1990a
1.5E–07	Liver	No data			U.S. EPA, 1990a
8.7E–08	Liver, kidney	Group D			U.S. EPA, 1990a
(k)		(k)			Dragun, 1988
1.9E–07	Liver	No data			U.S. EPA, 1989b
1.0E–07	Liver	7.5E–03[h]	7.0E–10	B2; liver	U.S. EPA, 1989b & 1990a
8.2E–06	Fetotoxic	Group D			U.S. EPA, 1989b & 1990a
(k)		(k)			Dragun, 1988
(k)		(k)			Dragun, 1988
6.8E–05	Liver	5.1E–02[h,m]	3.5E–08	B2[m]; liver	U.S. EPA, 1989b & 1990a
2.5E–08	Blood	Group D			U.S. EPA, 1989b & 1990a
8.7E–07	Liver	Group D			U.S. EPA, 1989b & 1990a
		1.7E–02[i,n]	1.3E–07	B2[n]; lung	U.S. EPA, 1989b
1.9E–06	Lung, heart	No data			U.S. EPA, 1989b
1.0E–08	Central nervous system	No data			U.S. EPA, 1989b & 1990a
1.9E–07	Central nervous system	Group D			U.S. EPA, 1989b & 1990a
2.6E–03		Total	1.9E–05		

Table 3-8. (Continued)

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in the text, the locations for samples SSS-009 and SSD-008 have been remediated.

[b]See Appendix N of the RI report (Thorpe *et al.*, 1990) for monitoring data for organic chemicals in soil samples from which maximum soil concentrations for the organic chemicals on the LLNL Livermore site were derived.

[c]According to the EPA weight-of-evidence classification system for carcinogenicity (U.S. EPA, 1990a), group A substances are human carcinogens; group B1 or B2 substances are probable human carcinogens (B1 indicates limited human data are available and B2 indicates sufficient evidence in animals and inadequate or no evidence in humans); group C substances are possible human carcinogens; and group D substances are not classifiable as to human carcinogenicity.

[d]Oral RfD (an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious noncarcinogenic effects during a lifetime) reported in IRIS data base (U.S. EPA, 1990a) or Fourth Quarter FY1989 Health Effects Assessment Summary Tables (U.S. EPA, 1989b); only an oral RfD was available.

[e]The RfD is based on an acceptable daily intake (ADI) for a newborn cited by Geyer *et al.* (1986).

[f]Considered to be the no-observed-adverse-effect-level (NOAEL).

[g]Evidence that PCBs, such as Aroclor 1254, are carcinogenic to humans is considered limited (IARC, 1982b).

[h]Oral carcinogenic-potency factor (CPF). Defined as the "slope factor" resulting from application of a low-dose extrapolation procedure.

[i]Inhalation CPF. Defined as the "slope factor" resulting from application of a low-dose extrapolation procedure.

[j]For trans-1,2-DCE isomer and applied to total 1,2-DCE.

[k]Naturally occurring organic chemical found in soil (Dragun, 1988) for which an inhalation RfD and a CPF have not been derived.

[l]Inhalation RfD (an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious noncarcinogenic effects during a lifetime) reported in IRIS data base (U.S. EPA, 1990a). Only an inhalation RfD was available in the IRIS data base.

[m]CPF and B2 classification are from U.S. EPA (1989b): *Fourth Quarter FY1989 Health Effects Assessment Summary Tables (HEAST)*; the CPF and weight of evidence grouping for PCE is "pending" in the IRIS data base (U.S. EPA, 1990a).

[n]CPF and B2 classification are from U.S. EPA (1989b): *Fourth Quarter FY1989 HEAST* document; the weight of evidence classification and CPF have been withdrawn from the IRIS data base (U.S. EPA, 1990a).

[o]Includes a value for $e_{2b\text{-}max}$(aos) equal to 2.4×10^{-6} mg/kg-d, which is based on a value for $C_{a\text{-}max}$(aos) equal to 4.6×10^{-5} mg/m^3 and a value for F_{2b}(aos) equal to 3.2×10^{-2} m^3/kg-d (see Appendix A).

First, the carcinogenic risk assessment presented in Table 3-12 is based on TTLC values [see data base in Appendix Table N-3 of the RI report (Thorpe *et al.*, 1990)], which do not take into account the mobility of inorganic chemicals in soil. The STLC values appearing in Appendix Table N-4 of the RI report (Thorpe *et al.*, 1990) provide a clearer picture of the likely mobility of such inorganic chemicals in soil. Nevertheless, STLC values certainly are not as conservative as TTLC values, and STLC values are given in units of milligram of substance per volume of leachate (e.g., typically mg/L) rather than milligram of substance per unit mass of soil (e.g., typically mg/kg).

Table 3-9. Frequency of detection [by total threshold limit concentration (TTLC) analysis] of inorganic chemicals at soil/sediment sampling locations on the LLNL Livermore site or on adjacent properties.

Chemical	Number of soil/sediment samples with detectable concentrations	Total number of soil/sediment samples	Percent detected
Antimony	9	57	15.8
Arsenic	57	57	100.0
Barium	57	57	100.0
Beryllium	40	57	70.2
Cadmium	31	57	54.4
Chromium	57	57	100.0
Cobalt	57	57	100.0
Copper	57	57	100.0
Lead	57	57	100.0
Mercury	26	57	45.6
Molybdenum	5	57	8.8
Nickel	49	49	100.0
Selenium	1	49	2.0
Silver	15	57	26.3
Thallium	3	49	6.1
Titanium	8	8	100.0
Vanadium	57	57	100.0
Zinc	57	57	100.0

Second, only total chromium was measured in the soil samples. Chromium (VI), which is the most carcinogenic species of this chemical and the basis for the CPF that is used (see Table 3-11), is presumed to be present only as a fraction of the total. Therefore, even though the maximum risk level associated with exposure to inorganic chemicals for adults onsite is attributed to a maximum concentration of total chromium of 1,500 mg/kg (i.e., Risk (R) = 1.2×10^{-1}), this level of risk may be far lower if the chromium (VI) is a very small fraction of the total concentration of chromium. Furthermore, the highest concentration onsite after the excavation and disposal of soil from sample sites SSD-008, SSD-009, and SSS-009 is only 110 mg/kg [see Appendix Table N-3 of the RI report (Thorpe *et al.*, 1990)].

Finally, the maximum soil concentrations for inorganic chemicals that appear in Table 3-12 (as well as in Tables 3-10 and 3-11) [$C_{s\text{-}max}^{inorg}$] do not exist at locations onsite where exposure by adults is likely to occur continuously for a hypothetical 50-y period. Sample locations were chosen in areas of sediment believed most likely to have accumulated contaminants for initial screening purposes, not to be representative of all soils onsite. Nevertheless, even after the

Table 3-10. Screening analyses for potential noncarcinogenic hazard resulting from soil-based exposures to inorganic chemicals by adults onsite for a continuous 50-y period.

Chemical	Location[a]	$[C_{s\text{-}max}^{inorg}]$ Maximum soil TTLC at LLNL Livermore site and adjacent locations (mg/kg)	Maximum total daily dose (all exposure pathways), $E_{s\text{-}max}^{inorg}$ (mg/kg-d)[b]	Oral reference dose, RfD (mg/kg-d)[c]	Hazard index for noncarcinogenic effects, HI $[E_{s\text{-}max}^{inorg}/RfD_{max}]$	Target[c]
Antimony	SSS-009	13	2.4E–05	4.0E-04	6.0E-02	Blood
Arsenic	SSS-003	14	2.6E–05	1.0E–03	2.6E–02	Skin
Beryllium	SSD-009	4	7.4E–06	5.0E–03	1.5E–03	Not determined
Cadmium	SSS-009	23	4.3E–05	5.0E–04	8.6E–02	Kidney
Chromium (total)	SSS-009	1,500	2.8E–03	5.0E–03[d]	5.6E–01	Reduced water consumption
Cobalt	ALPE	22	4.1E–05	1.6E+00[e]	2.6E–05	Not determined
Copper	SSS-009	530	9.8E–04	2.9E–02[f]	3.4E–02	Gastrointestinal tract
Lead	SSD-009	320	5.9E–04	1.4E–03[f]	4.2E–01	Central nervous system
Mercury	SSD-009	20	3.7E–05	3.0E–04	1.2E–01	Central nervous system
Molybdenum	SSS-009	16	3.0E–05	2.1E–03[g]	1.4E–02	Not determined
Nickel	SSS-009	670	1.2E–03	2.0E–02	6.0E–02	Reduced organ and body weights
Silver	SSS-009	7.4	1.4E–05	3.0E–03	4.7E–03	Brain
Zinc	SSS-009	750	1.4E–03	2.0E–01	7.0E–03	Blood
				Total	1.4E+00	

Table 3-10. (Continued)

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in the text, the locations for samples SSS-009 and SSD-009 have been remediated.

[b]See Appendix Table A-9.

[c]Only oral reference dose data available from U.S. EPA (1989b and/or 1990a), unless otherwise noted; target organ or effect of concern usually is identified in these references.

[d]Oral RfD for chromium (VI) is used (see IRIS data base; U.S. EPA, 1990a).

[e]Estimated ADI based on evidence reported in the literature that $CoSO_4$ has been used therapeutically at up to 300 mg/d without any symptoms of adverse health effects (National Research Council Safe Drinking Water Committee, 1977). Consequently, the ADI corresponds to a daily dose of cobalt of 114 mg/d divided by 70 kg.

[f]Estimated ADI based on the Federal and the State of California MCL for this chemical in drinking water (U.S. EPA, 1988a and d; and DHS, 1989a and d) and assuming a consumption rate of 2 L/d. Consequently, the ADI corresponds to the MCL multiplied by 2 L/d and divided by 70 kg.

[g]Estimated ADI based on the National Research Council Committee on Dietary Allowances (1980), which established 0.15 to 0.5 mg/d as an estimate of the safe and adequate intake range for molybdenum. To be conservative, the lower value was used, and the ADI corresponds to that value being divided by 70 kg.

Table 3-11. Screening analyses for potential noncarcinogenic hazard resulting from exposures to inorganic chemicals by adults onsite, arranged by target organ.

Chemical	Location[a]	$[C_{s\text{-}max}^{inorg}]$ Maximum soil TTLC at LLNL Livermore site and adjacent locations (mg/kg)	Maximum total daily dose (all exposure pathways), $E_{s\text{-}max}^{inorg}$ (mg/kg-d)[b]	Oral reference dose, RfD (mg/kg-d)[c]	Hazard index for noncarcinogenic effects, HI $[E_{s\text{-}max}^{inorg}/RfD_{max}]$	Target[c]
Antimony	SSS-009	13	2.4E–05	4.0E–04	6.0E–02	Blood
Zinc	SSS-009	750	1.4E–03	2.0E–01	7.0E–03	Blood
				Subtotal	6.7E–02	
Arsenic	SSS-003	14	2.6E–05	1.0E–03	2.6E–02	Skin
Beryllium	SSD-009	4	7.4E–06	5.0E–03	1.5E–03	Not determined
Cadmium	SSS-009	23	4.3E–05	5.0E–04	8.6E–02	Kidney
Chromium	SSS-009	1500	2.8E–03	5.0E–03[d]	5.6E–01	Reduced water consumption
Cobalt	ALPE	22	4.1E–05	1.6E+00[e]	2.6E–05	Not determined
Copper	SSS-009	530	9.8E–04	2.9E–02[f]	3.4E–02	Gastrointestinal tract
Nickel	SSS-009	670	1.2E–03	2.0E–02	6.0E–02	Reduced organ and body weights
Molybdenum	SSS-009	16	3.0E–05	2.1E–03[g]	1.4E–02	Not determined
				Subtotal	7.8E–01	
Lead	SSD-009	320	5.9E–04	1.4E–03[f]	4.2E–01	Central nervous system
Mercury	SSD-009	20	3.7E–05	3.0E–04	1.2E–01	Central nervous system
Silver	SSS-009	7.4	1.4E–05	3.0E–03	4.7E–03	Brain
				Subtotal	5.5E–01	
				Total	1.4E+00	

Table 3-11. (Continued)

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in the text, the locations for samples SSS-009 and SSD-009 have been remediated.

[b]See Appendix Table A-9.

[c]Only oral reference dose data available from U.S. EPA, 1989b and/or 1990a, unless otherwise noted; target organ or effect of concern usually is identified in these references.

[d]Oral RfD for chromium (VI) is used (see IRIS data base; U.S. EPA, 1990a).

[e]Estimated ADI based on evidence reported in the literature that $CoSO_4$ has been used therapeutically at up to 300 mg/d without any symptoms of adverse health effects (National Research Council Safe Drinking Water Committee, 1977). Consequently, the ADI corresponds to a daily dose of cobalt of 114 mg/d divided by 70 kg.

[f]Estimated ADI based on the Federal and the State of California MCL for this chemical in drinking water (U.S. EPA, 1988a and d; and DHS, 1989a and d) and assuming a consumption rate of 2 L/d. Consequently, the ADI corresponds to the MCL multiplied by 2 L/d and divided by 70 kg.

[g]Estimated ADI based on the National Research Council Committee on Dietary Allowances (1980) establishing 0.15 to 0.5 mg/d as an estimate of the safe and adequate intake range for molybdenum. To be conservative, the lower value was used and the ADI corresponds to that value being divided by 70 kg.

Table 3-12. Screening analyses for potential carcinogenic risk resulting from soil-based exposures to inorganic chemicals by adults onsite for a continuous 50-y period.

Chemical	Location[a]	$[C_{s\text{-}max}^{inorg}]$ Maximum soil TTLC at LLNL Livermore site and adjacent locations (mg/kg)	Maximum total daily dose (all exposure pathways), $E_{s\text{-}max}^{inorg}$ (mg/kg-d)[b]	Weight-of-evidence (group)[c]; Target[d]	(CPF_{max}) cancer potency factor (1/[mg/kg-d])[d]	$[E_{s\text{-}max}^{inorg}) \bullet CPF_{max}]$ Risk related to carcinogenic effects (R)
Antimony	SSS-009	13	2.4E–05	Noncarcinogen		
Arsenic	SSS-003	14	2.6E–05	A; lung & skin	1.5E+01[e]	3.9E–04
Beryllium	SSD-009	4	7.4E–06	B2; lung & bone	8.4E+00[e]	6.2E–05
Cadmium	SSS-009	23	4.3E–05	B1; kidney	6.3E+00[e]	2.7E–04
Chromium (total)	SSS-009	1,500	2.8E–03	A; lung	4.2E+01[f]	1.2E–01
Cobalt	ALPE	22	4.1E–05	No data		
Copper	SSS-009	530	9.8E–04	D; noncarcinogen		
Lead	SSD-009	320	5.9E–04	B2; kidney	Not determined	
Mercury	SSD-009	20	3.7E–05	D; noncarcinogen		
Molybdenum	SSS-009	16	3.0E–05	No data		
Nickel	SSS-009	670	1.2E–03	A; respiratory tract	8.4E–01[e]	1.0E–03
Silver	SSS-009	7.4	1.4E–05	D; noncarcinogen		
Zinc	SSS-009	750	1.4E–03	No data		
					Total	1.2E–01

[a]As noted in the text, the locations for samples SSS-009 and SSD-009 have been remediated.

[b]See Appendix Table A-9.

[c]According to the EPA weight-of-evidence classification system for carcinogenicity (U.S. EPA, 1990a), group A substances are human carcinogens; group B1 or B2 substances are probable human carcinogens (B1 indicates limited human data are available and B2 indicates sufficient evidence in animals and inadequate or no evidence in humans); group C substances are possible human carcinogens; and group D substances are not classifiable as to human carcinogenicity.

[d]Data from U.S. EPA, 1989b and/or 1990a.

[e]CPF for inhalation.

[f]Only total chromium was measured in soil samples, but CPF is for chromium (VI) , and chromium (VI) is presumed to be only a fraction of the total.

excavation and removal of the sediment at sites SSD-008/SSD-009 and SSS-009, the soil at location SSS-003 (Fig. 3-13) poses a screening level maximum risk of 3.9×10^{-4} as a consequence of an elevated arsenic concentration and arsenic's substantial CPF value. These sediments and soils are considered to be candidates for excavation and disposal. Consequently, by excavating and safely disposing of the sediments and soils from identified locations, the potentially high cancer risk predicted in Table 3-12 has already been substantially lowered and will be lowered further by simple removal actions. (The same is true for risks from exposure to organic chemicals also detected at these locations, see Table 3-8). Moreover, administrative controls are in place to prevent any significant occupational exposure to elevated concentrations of inorganic chemicals in soil from occurring. Additional soil sampling will be performed to determine if monitored TTLC and STLC concentrations of inorganic chemicals in soil are the result of an old source that has been cleaned up or are the result of an existing source that requires remediation. Appendix B contains tables of exposures and associated hazards and risks for inorganic chemicals computed using typical EPA methodology and parameters.

As mentioned earlier, we included the concentrations of inorganic chemicals in the soils sampled from the arroyos adjacent to the LLNL Livermore site (locations SAS-011 and SAP-001 in Fig. 3-12) in our analyses of the soil sampling data that was performed using the procedure of Michels (1971, Appendix C). We can now determine if the concentrations of inorganic chemicals in the soils sampled from the arroyos differ from background levels. The results of this analysis (C-1 through C-17 in Appendix C) indicate that no clear distinction can be made between the concentrations of inorganic chemicals in the soil samples from the arroyos and background levels. Even the maximum concentrations of nickel and silver in the soil samples taken from the arroyos (i.e., 32 and 0.9 mg/kg, respectively), which approach the maximum of the range of concentrations reported for LLNL Site 300 and exceed the range of concentrations for selected California counties (Appendix Table C-1), are not considered to be part of a distribution that includes increments from a local source and that could be considered distinct from the background distribution. Consequently, inorganic chemicals in the soils of the arroyos adjacent to the site are not considered to pose a health hazard to the public offsite.

3.2.4.4. Radioactivity

Monitoring data for radioactive substances above the LOD in the surface soils of the LLNL Livermore site, adjacent properties, and the Livermore-Amador Valley area (LLNL, 1990; Brekke *et al.*, 1989) are presented in Appendix Tables N-5 through N-7 of the RI report (Thorpe *et al.*, 1990). For those radioactive substances detected on the LLNL Livermore site or adjacent properties (LLNL, 1990; Brekke *et al.*, 1989) and for which concentration data have been reported for surface soils in the Livermore-Amador Valley area (Brekke *et al.*, 1989)—cesium–137 (^{137}Cs), plutonium–239+240 ($^{239+240}$Pu), potassium–40 (^{40}K), thorium–232 (^{232}Th), tritium (^{3}H), and uranium–238 (^{238}U)—we again used the procedure of Michels (1971, Appendix C) to determine if any of the concentrations in the surface soils sampled differ from background levels. The results of this analysis (Figs. C-18 through C-23 in Appendix C) indicate that no distinction can be made between the maximum concentrations of $^{239+240}$Pu (that are not "outliers" as noted by Brekke *et al.*, 1989), ^{40}K, ^{232}Th, and ^{238}U in the soil samples from the arroyos and background levels. However, such a distinction can be made with regard to ^{137}Cs and ^{3}H.

Only gross beta activity at a maximum concentration of 32 pCi/g was detected in soils sampled from the arroyos near the LLNL Livermore site [location SAP-001; see Fig. 3-12 and data in Appendix Table N-5 of the RI report (Thorpe *et al.*, 1990)]. Accordingly, additional soil sampling will be performed to determine if the ^{137}Cs and ^{3}H concentrations in soil samples onsite and the gross beta activity monitored in the arroyo offsite (> LOD) are attributable to a source onsite, and if so, whether that source requires remediation. Also, administrative controls are in place to prevent adults onsite from working continuously in the areas identified as having elevated levels of the aforementioned radioactive substances. Furthermore, the soils with the highest concentrations of radioactive substances were at site SSD-008/SSD-009, and the surficial soils from these locations have been removed.

Because two of the most important radioactive substances for which concentrations were determined in the sampling studies are tritium and $^{239+240}Pu$, we evaluated the maximum soil concentrations reported for these substances further. The highest concentration of tritium in soils (activity in recovered soil water) detected onsite was 11,000 pCi/L (in Appendix Table N-5 of the RI report (Thorpe *et al.*, 1990). The maximum concentration of $^{239+240}Pu$ in soil was less than 21 pCi/kg [Appendix Tables N-5 through N-7 of the RI report (Thorpe *et al.*, 1990)]. The maximum concentration for $^{239+240}Pu$ in the soil, which has an estimated bulk density of 1,500 kg/m^3, is more than two orders of magnitude (100 times) lower than the U.S. EPA (1977) interim recommendations for a soil with this bulk density (13,000 pCi/kg).

The concentrations of tritium and ^{239}Pu detected in the soils in the most recent sampling studies are not of concern from the perspective of human exposure. Furthermore, as tritium undergoes radioactive decay, the concentration will decline. Concentrations of tritium were below the LOD (i.e., < 1,000 pCi/L) in soils sampled in the arroyos offsite (LLNL, 1990). As mentioned previously, the other radioactive substances, except ^{137}Cs, that were monitored in soils were at background levels. In fact, radioactivity on air filters on the LLNL Livermore site perimeter for a variety of different radionuclides, including ^{137}Cs, ^{40}K, ^{226}Ra, ^{228}Ra, ^{228}Th, $^{239+240}Pu$, ^{238}U, and tritium, were well below the derived concentration guide (DCG) established by the DOE (Brekke *et al.*, 1989). These data indicate that LLNL is not emitting significant concentrations of these radioactive substances into the air, which might be deposited on surface soil. On the basis of the assessment just presented, we conclude that radioactive substances in soils onsite and offsite do not constitute a problem with regard to human exposure.

3.2.5. Summary

Table 3-13 summarizes the types and estimated amounts of VOCs found in ground water in the study area. Our review of the concentration data on the various organic and inorganic constituents of ground water indicates that the dominant substances of concern are TCE, PCE, and chloroform. These VOCs are widely distributed across the site, constitute an estimated 91% of the total mass of the VOCs, and are classified as B2 carcinogens (see also Section 5.1.2.1). [According to the EPA Weight-of-Evidence Classification System for Carcinogenicity, "a Group B2 Carcinogen is described as a probable human carcinogen indicated by sufficient evidence in animals and inadequate or no evidence in humans" (U.S. EPA, 1989a).] The transport and transformation of TCE, PCE, and chloroform in ground water are addressed in Section 4 of this document.

Table 3-13. Summary of estimated masses and volumes of VOCs in ground water.

Compound	Density (kg/L)	Mass (kg)	Percent total mass	Volume (L)	Volume (gal)
TCE	1.46	603	64	412	109
PCE	1.62	196	21	121	32
Chloroform	1.48	61	6	90	24
Total		860	91	623	165
"Other VOCs":					
1,1-DCE	1.22	47	5	39	10
1,2-DCE (total)	1.27	5	0.5	4	1
Carbon tetrachloride	1.59	9	1	6	2
1,1,1-TCA	1.34	5	0.5	4	1
1,1-DCA	1.18	9	1	8	2
1,2-DCA	1.24	9	1	7	2
Total "other VOCs"		84	9	68	18
Total VOCs		944	100	691	183

The other VOCs in ground water constitute the remaining 9% of the mass and are apportioned as indicated in Table 3-13. The compound 1,1-DCE accounts for 56% of the "other VOCs" category. We use carbon tetrachloride in the following section, as the indicator compound to assess the health risk of the remaining 44% of the "other VOCs" category because it has the highest cancer potency of this group. However, carbon tetrachloride is not present in all the contaminant plumes that contain 1,1-DCE and the other compounds in this category. Most notably, carbon tetrachloride is not a constituent of the offsite plume in the Southwest Area. We take this into account in the following section when estimating the health risk-associated with carbon tetrachloride and the "other VOCs."

Our evaluation of the monitoring data for the organic and inorganic chemicals in surficial soil and for radioactivity in surficial soil indicates that the concentrations of these substances onsite and offsite are not sufficient to pose a noncarcinogenic health hazard or a carcinogenic health risk by any exposure pathway. This evaluation is based on the application of pathway exposure factors and the calculation of hazard indices for noncarcinogenic effects and risks related to carcinogenic effects using a method similar to that described by the U.S. EPA (1986a).

Section 3 References

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4. Contaminant Migration

M. C. Small, P. F. McKereghan, D. W. Layton, E. M. Nichols, R. K. Thorpe,
M. D. Dresen, A. F. B. Tompson, C. H. Hall, F. Yukic, and K. Goyal

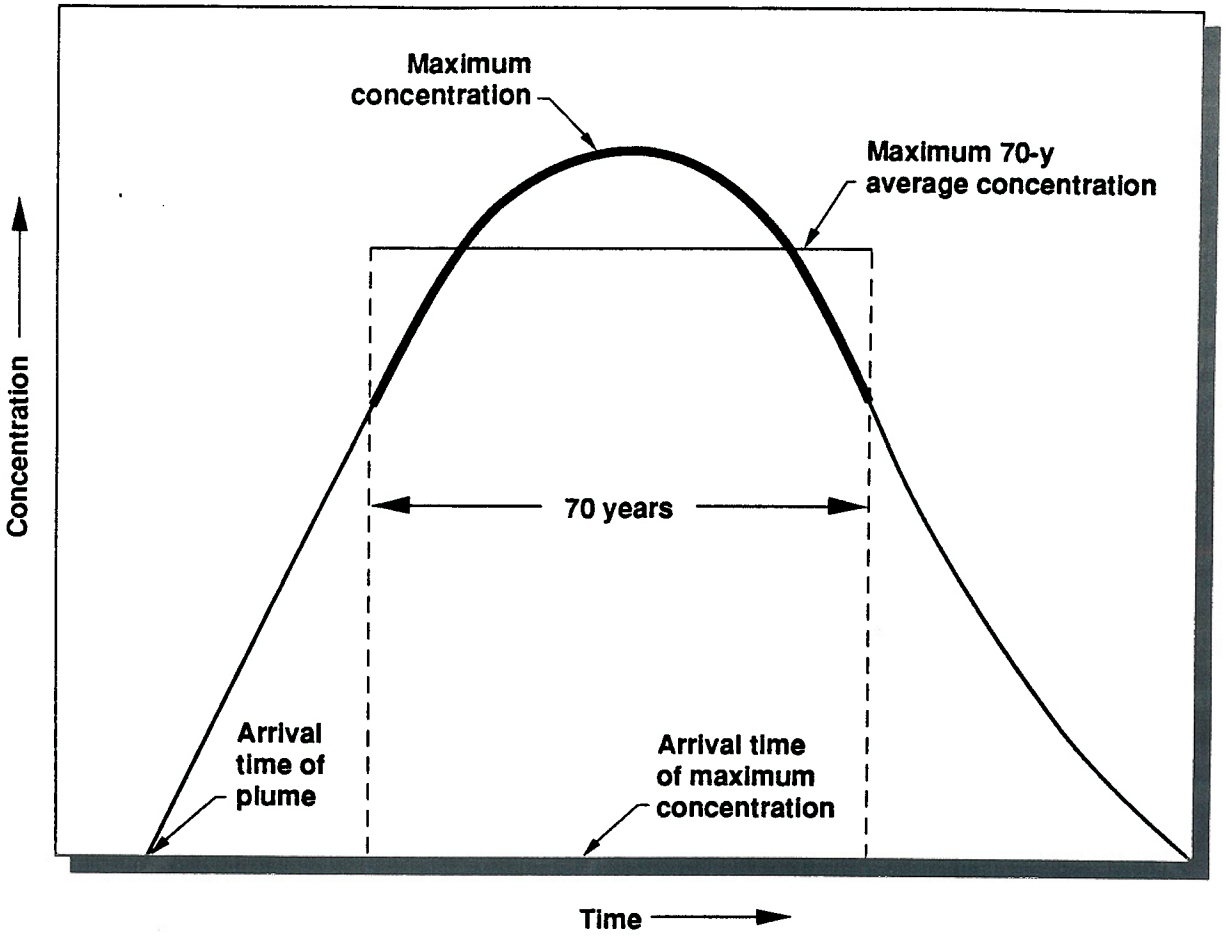
4.1. Objective

In this section, we estimate contaminant concentrations that might occur in existing or potential offsite water supply wells in the future, assuming the contaminants we identified earlier will be transported by ground water. The objective is to calculate an individual's potential maximum, continuous 70-y exposure to contaminated well water, as diagrammed in Figure 4-1. This 70-y exposure period will be used in Section 5 of this document to assess the public health risk. As previously noted, because this is a baseline assessment, we assume that there are no efforts to remediate contaminants in ground water or to avert exposures by providing substitute water supplies. However, LLNL is already in the process of pilot remediation and is committed to continued cleanup efforts.

The major contaminant plumes have been characterized in Section 4 of the RI report (Thorpe *et al.*, 1990). However, there remains some uncertainty as to the future rate and direction of migration beyond their present positions. In practice, this can be predicted (1) by empirical means (e.g., tracking natural ground water tracers) or (2) by using a calculational model to simulate the transport and transformation process over the flow domain (i.e., between source(s) and receptors). To account for uncertainty in direction, we make the conservative assumption that all ground water flows westward, directly toward the potential receptor wells. This justifies use of a relatively simple flow and transport model, described in detail in this section.

The hydraulic gradient and properties of the geologic medium are assumed to be constant over this domain for any given simulation. The variation of input parameters among different simulations represents uncertainty in the rate of contaminant migration. To bound this uncertainty, we use two sets of parameters, consisting of "best-estimate" and "health-conservative" values. The best-estimate parameters are values that are the most likely or representative, based on our current knowledge of the ground water system and contaminant properties. The health-conservative set consists of extreme values that yield exposures that are unlikely to be exceeded.

More sophisticated numerical models will be utilized for the FS and later remedial action phases to design and evaluate cleanup alternatives. These models will provide more detailed simulations of the hydrogeology and physical processes on both regional and local scales. The process of formulating and implementing these models is currently in progress and will be fully described in later documents.



ERD-LSP-91-0016

Figure 4-1. Generalized depiction of time-varying concentration of a contaminant in a well showing data used to calculate maximum 70-y exposure.

4.2. Model Domain

4.2.1. Hydrogeologic Summary

As discussed in more detail in Section 3 of the RI report (Thorpe *et al.*, 1990), the sediments beneath the study area constitute two water-bearing systems: the upper system, composed of Recent alluvium underlain by virtually identical sediments of the Upper Member of the Livermore Formation, and the lower system, consisting of alluvial and lacustrine sediments of the Lower Member of the Livermore Formation. The permeable sediments in these two systems are separated by the horizontally extensive, low-permeability confining layer that occurs near the top of the Lower Member of the Livermore Formation. Interconnected permeable sediments, consisting of buried stream-channel and overbank deposits within these units, constitute preferred flow pathways for migration of ground water and contaminants.

The Upper Livermore Formation beneath the LLNL site and adjacent areas consists of complexly interfingered alluvial sediments of highly variable permeability and thickness (Carpenter *et al.*, 1984). The more permeable deposits vary in thickness from less than 1 m to 10 m. The less permeable deposits are commonly 1 to 7 m thick, but are locally as thick as 25 m. This description is typical of heterogeneous alluvial deposits in which individual small channels are difficult, if not impossible, to define by exploratory boring. Consequently, common practice is to represent the subsurface as an equivalent homogeneous system. This simplification is admissible because the scale of observation (kilometers) is much larger than the scale of heterogeneities of the interconnected permeable sediments (1 to 100 m).

Depth to water in the LLNL site area is about 40 m in the southeast corner of the LLNL site, about 20 m at the western boundary of the site, and about 10 m in the vicinity of downtown Livermore. Combined with geomorphic and topographic information, hydraulic test data indicate that the primary directions of streams that deposited the LLNL site sediments varied from westward to northwestward (Dresen and Hoffman, 1986). This is generally the same direction that surface waters follow today. The present patterns of contaminants in ground water also appear to display this orientation. Based on analysis of geologic cross sections and interpretation of pumping test data [Sections 3.4 and 3.6, respectively, of the RI report (Thorpe *et al.*, 1990)], some of the higher permeability sediments are in relatively good horizontal hydraulic communication, whereas less interconnection exists between layers vertically. This supports a single-aquifer model, as described below.

Little or no hydraulic communication exists between the sediments of the upper unit and the permeable deposits of the Lower Member of the Livermore Formation, due to the presence of the intervening confining layer comprising the upper 10 to 30 m of the Lower Member of the Livermore Formation. A 2.1 m^3/min pumping test conducted on former agricultural well 13D1, completed in and above the confining layer, showed no communication with well 12N1, which was 230 m away and was completed below the confining layer. Substantial communication was observed in many upper unit wells at distances up to 1,220 m (Dresen *et al.*, 1988). Other pumping tests on upper-unit monitor wells have shown little or no communication with wells screened in or below the confining layer (Webster-Scholten *et al.*, 1987). Correspondingly, little or no VOCs have been detected within or beneath the confining layer. The hydraulic tests show that the sediments within the upper unit system are in hydraulic communication horizontally and, to a lesser extent, vertically, but they do not communicate with sediments within either the confining layer or the lower unit.

Although the geology of the study area is heterogeneous, it can be represented as an equivalent homogeneous system in the ground water flow model provided the model domain is much larger than the scale of the heterogeneities. The modeling domain includes the LLNL site and the area west (downgradient) to central Livermore, an area of about 11 km^2 (4 mi^2); whereas the heterogeneities are on the scale of meters. In our analysis, we account for the highest permeabilities, which lead to the shortest possible ground water travel time. The conceptual hydrogeologic model thus consists of a uniform effective ground water velocity toward the west, confined conditions above and below the contaminated aquifer, and a vertically uniform distribution of contaminants within the aquifer.

4.2.2. Current Ground Water Usage

The individuals potentially risking exposure to the contaminants are users of water from domestic and municipal supply wells west of the LLNL site, in the approximate downgradient ground water flow direction. These wells are shown in Figure 4-2 and described in Table 4-1. Most are used for domestic or irrigation purposes. The domestic wells still in service are generally well out of the expected paths of the existing contaminant plumes, but nonetheless are tested on a quarterly basis for contamination. If contaminants were detected in any of these wells, substitute water supplies would be provided, or the water would be treated before use. At present, none of these supply wells are contaminated.

Water for residential, commercial, and agricultural areas adjacent to the LLNL site comes from four sources:

- Private wells.
- Alameda County Flood Control and Water Conservation District (Zone 7).
- City of Livermore wells.
- California Water Service Company (CWSC) wells.

The water service areas for the City of Livermore and CWSC are shown in Figure 4-3.

Zone 7 serves as a wholesaler of water conveyed from the Sacramento–San Joaquin River Delta to the Livermore area via the South Bay Aqueduct System (SBAS). This source comprises about 75% of the total water distributed by Zone 7; the remainder is ground water. The delta water is treated and distributed continually, and only an incidental quantity is used for local ground water recharge. Thus, it is, in effect, blended in the Zone 7 distribution system; however, the degree of blending varies with location and time.

The City of Livermore purchases all its water from Zone 7. CWSC buys approximately 75% of its water from Zone 7; some of the remainder is supplied by wells 9L1, 9P1, 9Q1, 16B1, and 16C1 (Fig. 4-2). The CWSC wells near the center of Livermore serve 4,243 residences and businesses, representing about 9,500 people (Ekstrom, 1989). Some of the water from CWSC wells is blended with Zone 7 water before distribution.

The main agricultural water user in the vicinity of the LLNL site is the Wente Bros. Winery. The primary source of irrigation water for the vineyards directly south of East Avenue is surface water from Zone 7, augmented by ground water pumped from well 14C3 during the periods of highest water demand. This well water is introduced directly into the irrigation distribution system in response to a pressure-sensitive valve, and is not used for domestic consumption.

4.2.3. Future Ground Water Usage

It is important to assess whether new private wells are likely to be drilled in areas in the path of the migrating ground water plumes. LLNL has no ground water supply wells onsite, nor are there any plans for such. As discussed earlier in the context of the Federal Facility Agreement

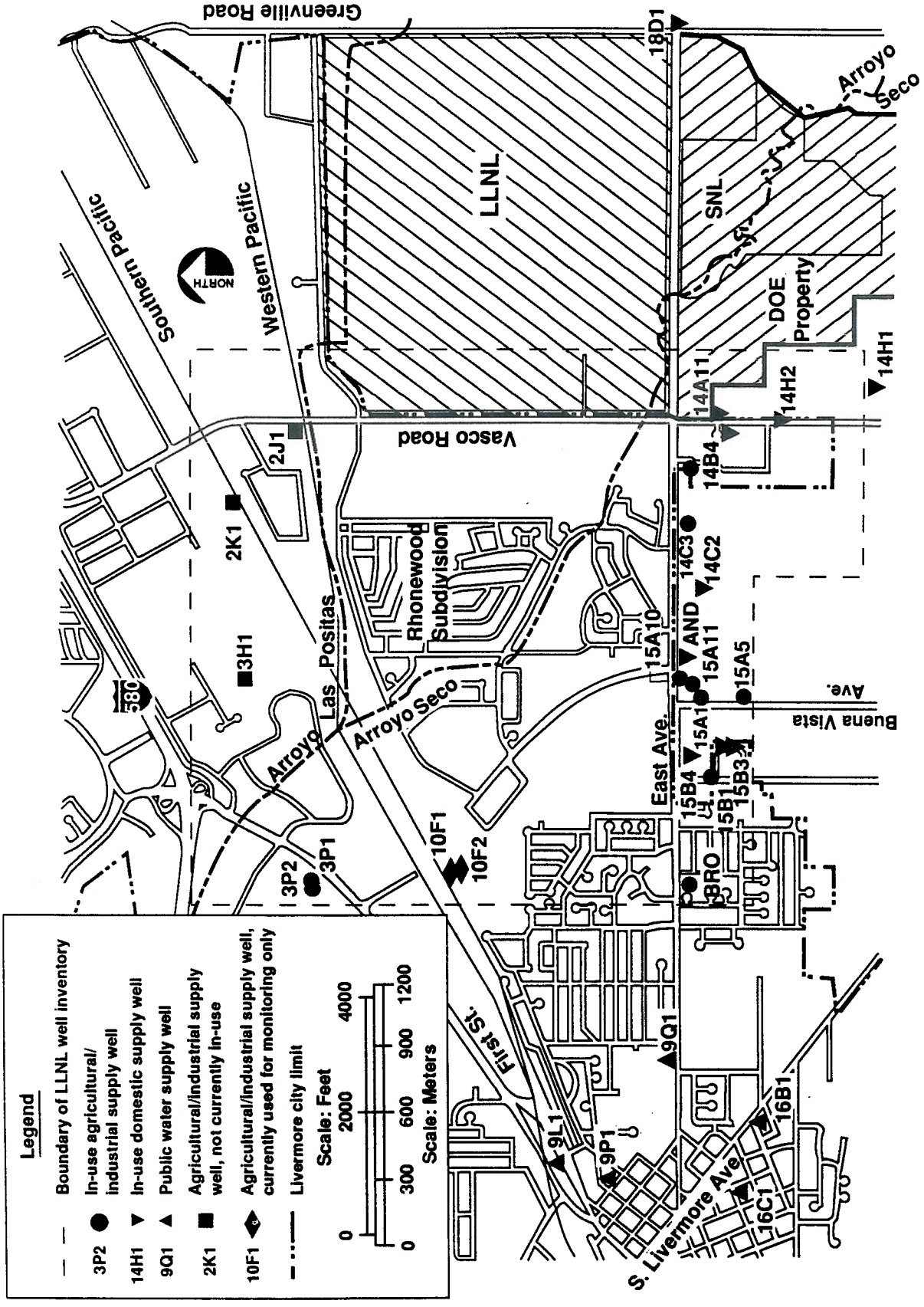


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Figure 4-2. Supply wells investigated during LLNL well inventories. (See Table 4-1 for well descriptions.)

Table 4-1. Public and private wells currently in operation in the LLNL vicinity that were investigated during the LLNL inventory. (See Fig. 4-2 for well locations.)

State well name	Flow rate (gpm)	Owner	Use	Comments
2J1	NA	Salinas Reinforcing	Irrigation/industrial[a]	Not currently in use
2K1	NA	Capital Metals	Industrial[b]	Not currently in use
3H1	NA	PG&E	Irrigation[a]	Not currently in use
3P1	NA	NA	Landscape irrigation[a]	
3P2	NA	Layton	Landscape irrigation[a]	
9L1, 9P1, 9Q1	>300[c]	CA Water Service	Public water supply wells[b]	Free of VOCs
10F1	>100[c]	Hexcel Corp.	Industrial[b]	Used for monitoring only
10F2	>100[c]	Hexcel Corp.	Industrial[b]	Used for monitoring only
14A11	NA	Phillips	Domestic[d]	Free of VOCs[e]
14B1	>25	Bargman	Swimming pool[d]	Free of VOCs, city water for domestic use
14B4	NA	Speral	Domestic[d]	Free of VOCs[e]
14C2	10[g]	Wente Bros.	Domestic[d]	Free of VOCs[e,f]
14C3	100[g]	Wente Bros.	Crop irrigation[d]	Free of VOCs[e]
14H1	NA	Miller	Domestic[d]	Free of VOCs[e]
14H2	NA	Freyendal	Domestic[d]	Free of VOCs[e]
15A1	5[g]	Casen	Landscape irrigation[a]	Temporarily out of service
15A5	NA	McGowen	Landscape irrigation[a]	
15A10	>10[g]	Livermore Preschool	Landscape irrigation[a]	
15A11	NA	Fitzgerald	Landscape irrigation[a]	Temporarily out of service
15B1	40[g]	Leeds	Landscape irrigation[a]	
15B2, 15B3	30–50[g]	Almond Circle Homeowners Assoc.	Landscape irrigation, swimming pool, and drinking water[a]	Very limited domestic use
15B4	NA	Mena	Domestic supply[a]	
16B1, 16C1	>200[c]	CA Water Service	Public water supply wells[b]	
18D1	NA	Williams	Domestic[d]	Free of VOCs[e]
AND[h]	10[g]	Anderson	Domestic supply[i]	Free of VOCs
BRO[h]	40[g]	Broadman	Landscape irrigation, livestock, and garden[j]	Free of VOCs

Table 4-1. (Continued)

[a]Not sampled for VOCs. Well is located more than 1/4 mi from LLNL VOC plume.

[b]Sampled for VOCs by owner. Well is located more than 1/4 mi from LLNL VOC plume.

[c]From current owner/user.

[d]Sampled during LLNL quarterly monitoring. Well is located within 1/4 mi of VOC plume.

[e]LLNL monitoring shows that this well is free of VOCs. [See Appendix D of the RI report (Thorpe *et al.*, 1990)].

[f]Well not currently in use: Wente Bros. planned to reactivate it in 1990.

[g]Based on existing records of Alameda County Flood Control and Water Conservation District, Zone 7.

[h]No existing State well name.

[i]Sampled for VOCs by LLNL on 24 May 1989. Well is located more than 1/4 mi from LLNL VOC plume. Sample does not appear in Appendix D of the RI report (Thorpe *et al.*, 1990).

[j]Sampled for VOCs by LLNL in 1984. Well is located more than 1/4 mi from LLNL VOC plume. Sample data does not appear in Appendix D of the RI report (Thorpe *et al.*, 1990).

NA = Information not available.

(U.S. EPA *et al.*, 1988), the continued use of the site for noncommercial, nonresidential use is a certainty. New offsite wells are unlikely for the following additional reasons:

- All subdivisions and commercial areas near the site are supplied by municipal water. Consequently, there is no compelling need for local residences to install new wells. In fact, some of the new residential developments have restrictions against private well construction.
- Ground water near the site is not a desirable source of drinking water because the total dissolved solids are over the State of California secondary standard of 500 mg/L (DHS, 1989).
- High nitrate levels are found in places throughout the Livermore Valley (Sorenson *et al.*, 1985).
- Levels of naturally occurring boron, a phytotoxic substance, are also high, which reduces the desirability of local ground water for irrigation.

Nevertheless, a land owner legally could have a well installed, provided that the necessary permit is obtained from Zone 7. According to Alameda County Ordinance 7368, Zone 7 is primarily responsible for protecting and managing local water resources. To protect ground waters from surface and subsurface pollution, Zone 7 has established criteria governing the installation and development of wells in the Livermore Valley. Thus, there is little likelihood of allowing a well to be completed in a contaminated zone.

The cost of completing a 60-m-deep well in the eastern Livermore Valley that meets Zone 7 requirements is about $10,000, which would effectively discourage most residents from installing a well. However, Wente Bros. Winery plans to reactivate well 14C2 and develop additional ground water sources for irrigation only. Possible exposure from this type of well is included in the risk assessment in Section 6. Any further wells in the area should be constructed in a manner that minimizes the possibility of encountering contamination or causing cross-contamination between water-bearing zones.

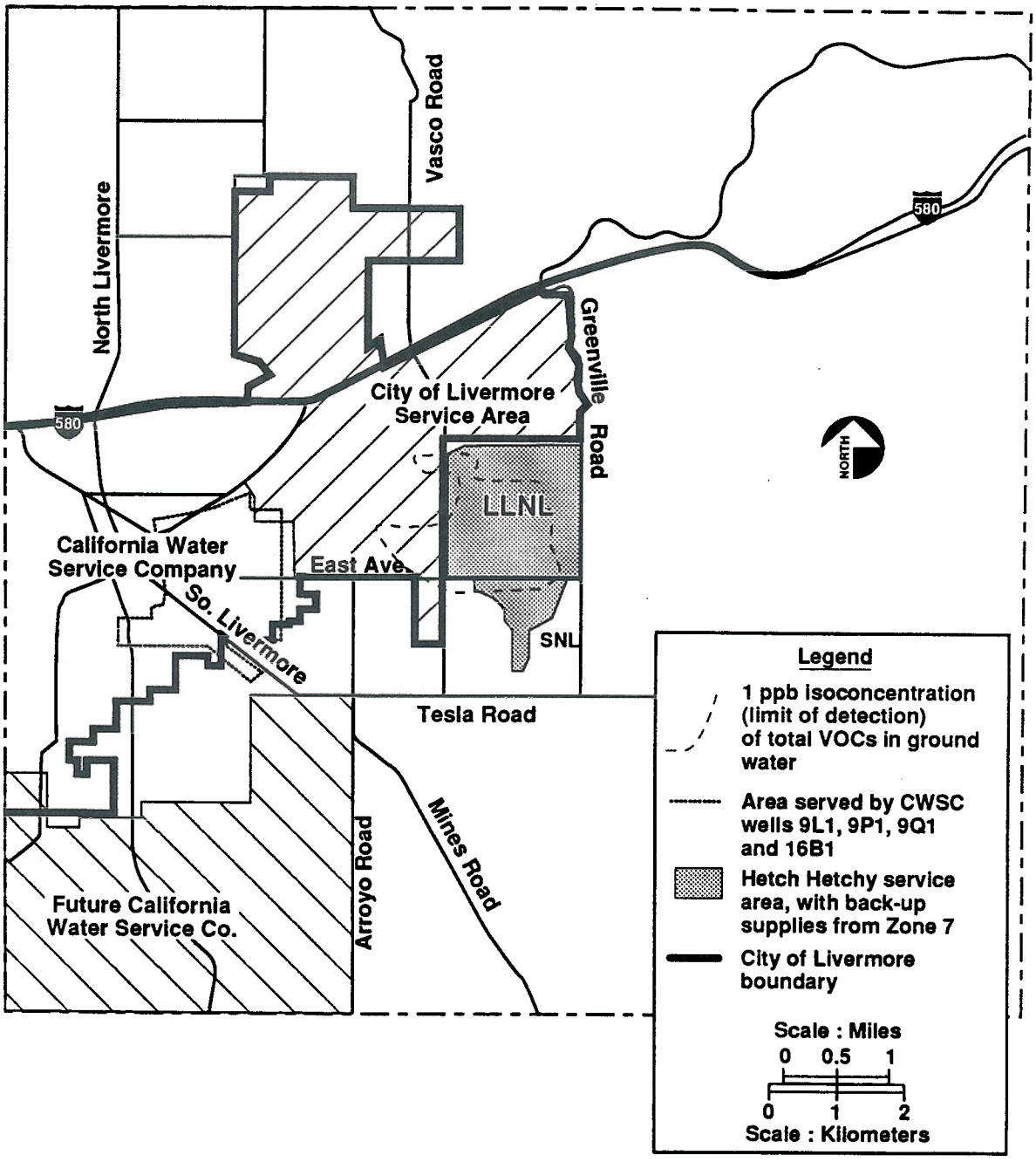

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Figure 4-3. Water service areas in a 220-km^2 (85-mi^2) area surrounding LLNL.

4.2.4. Receptor Locations

Potential receptors are located in the region west of the LLNL site and east of central Livermore. This area comprises the Mocho and southern portion of the Spring subbasins, as shown in Figure 4-4. The Mocho subbasin is divided into two ground water provinces, the eastern Mocho I province, which includes part of the LLNL site, and the western Mocho II province, which includes central Livermore. The degree of hydraulic communication between ground waters of the two provinces is somewhat uncertain, as evidenced by the very low and irregular potentiometric gradient between them. For this risk assessment, we have conservatively assumed that all of the shallow ground water at the LLNL site flows west to central Livermore. The locations of the potential receptor wells fall within this assumed path.

For calculational purposes, we have chosen observation points that represent existing and potential receptor wells directly west of the highest concentrations in each contaminant plume. By locating the observation points in this manner, rather than using actual well locations, we assume the maximum concentrations of each plume will intersect a receptor over the course of its westward migration. This is equivalent to making the conservative assumption that the VOC plumes could migrate directly toward the actual wells. As a result, the highest possible peak concentrations and greatest 70-y average concentrations are predicted at these locations.

Observation points for migration calculations are shown in Figure 4-5. We group them into three sets according to distance from the sources: near-field, mid-field, and far-field (denoted N, M, or F, respectively, in the figure). The near-field group represents potential monitor wells, from which water could be drawn and used domestically under purely hypothetical circumstances.

The far-field points represent existing municipal supply wells in central Livermore. The mid-field group is equidistant between the near- and far-field groups. Within each of these three groups, observation points are labeled A, B, and C from north to south. The A and C points are aligned directly west of the maximum concentrations of chloroform and PCE, respectively, in the existing plumes. The B points are west of the maximum concentrations for TCE and "other VOCs," represented by 1,1-DCE. A fourth set of observation points, labeled D-1, D-2, and D-3 in Figure 4-5, lies south of East Avenue and represents local domestic or agricultural supply wells not directly in the westward path of the plumes.

Under the health-conservative (most health-protective) scenario, all potential monitor wells in the near field (group N) are assumed to receive water only from contaminated zones (i.e., no dilution with ground water from cleaner zones occurs). Thus, the predicted concentrations for the A-N, B-N, and C-N observation points represent upper bounds. Moreover, these hypothetical wells would experience the most immediate contamination because they would be adjacent to the western site boundary. Such a situation, although highly improbable, has been assumed for a "worst case" in previous CERCLA risk assessments. We feel that this set of receptors represents the most stringent, yet plausible conditions for our health-conservative scenario.

On the other hand, agricultural and domestic supply wells typically draw water from many different zones over a depth much greater than the thickness of the existing contaminant plumes. Thus, observation points in a best-estimate scenario represent wells in which this type of dilution would occur. While we do not explicitly account for dilution in the following calculations, it is considered, where appropriate, in the exposure and risk assessments in Section 5.

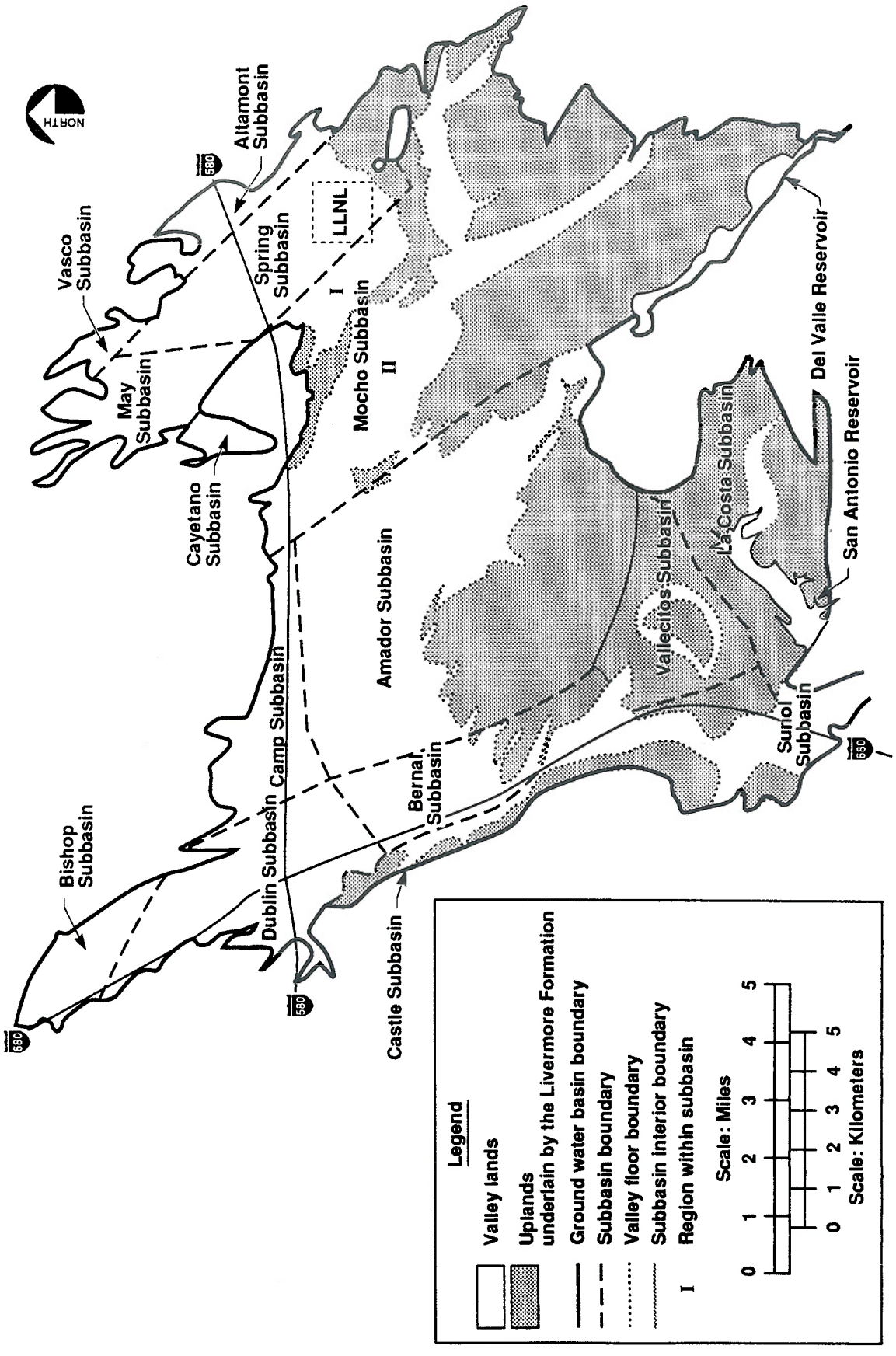

Figure 4-4. Location of subbasins and physiographic features of the Livermore Valley.

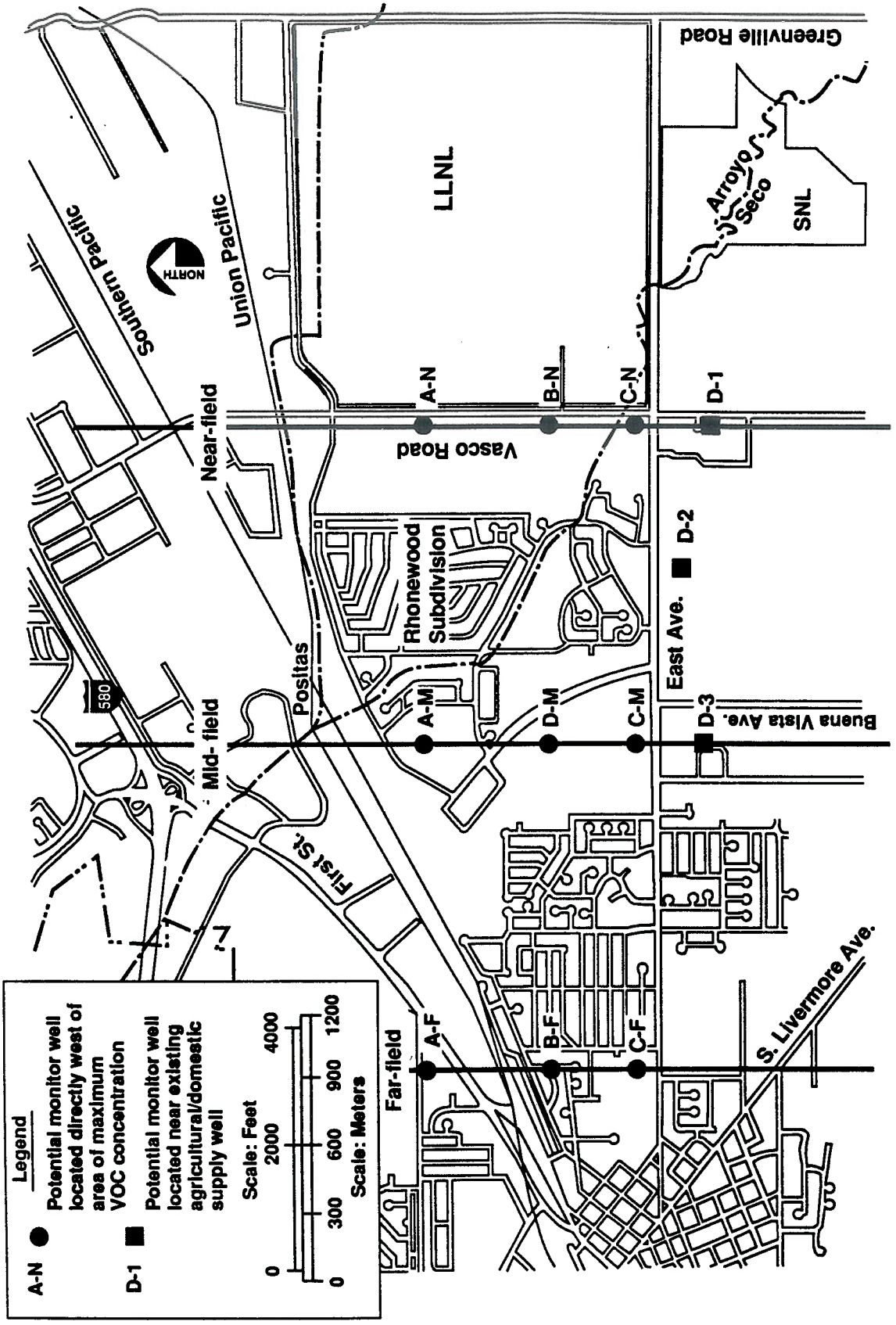

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Figure 4-5. Observation points representing existing and potential well locations for contaminant migration simulations.

4.3. Analytical Transport and Transformation Modeling

4.3.1. The PLUME Code

We used the analytical code PLUME (In-Situ, 1986) to calculate the transport and transformation of contaminants from LLNL to the observation points described above. The model is written in FORTRAN and BASIC for use on an IBM PC or compatible microcomputer. PLUME is an analytical solution of the general two-dimensional contaminant transport equation for a one-dimensional flow field:

$$D_L\frac{\partial^2 C}{\partial x^2} + D_T\frac{\partial^2 C}{\partial y^2} - v_x\frac{\partial C}{\partial x} = \left(1 + \rho_b\frac{K_d}{n}\right)\frac{\partial C}{\partial t} + kC\,, \qquad (4\text{-}1)$$

where

D_L and D_T = dispersion coefficients in the longitudinal and transverse directions, respectively;

v_x = seepage velocity in the x direction,

C = contaminant concentration,

t = time,

ρ_b = bulk density,

K_d = distribution coefficient,

n = porosity, and

k = first-order decay rate.

This equation includes the four processes involved in ground water transport and contaminant transformation. The primary transport mechanism is advection, the movement of solute with the average ground water velocity, and is represented by the third term on the left. The first and second terms represent hydrodynamic dispersion, consisting of mechanical spreading and molecular diffusion in two dimensions. The fourth term represents adsorption and desorption of contaminants, which retard the net rate of migration. The fifth term represents biological and/or abiotic transformation (degradation), both of which may alter or eliminate the compounds over time.

Figure 4-6 depicts the various inputs to the PLUME code. The aquifer is assumed to be homogeneous and isotropic, with constant thickness and porosity. We also assume that the ground water flows horizontally in only one direction, with constant velocity (steady-state conditions), and that dispersion occurs both longitudinally and transversely to the flow. Solute degradation is modeled as a first-order decay process with a constant transformation rate. Retardation of a solute, resulting from reversible sorption to subsurface materials, is calculated from a distribution coefficient (K_d) for the modeled contaminant and the bulk density and porosity of the subsurface materials. These assumptions are admissible and appropriate for the hydrogeologic domain of this study.

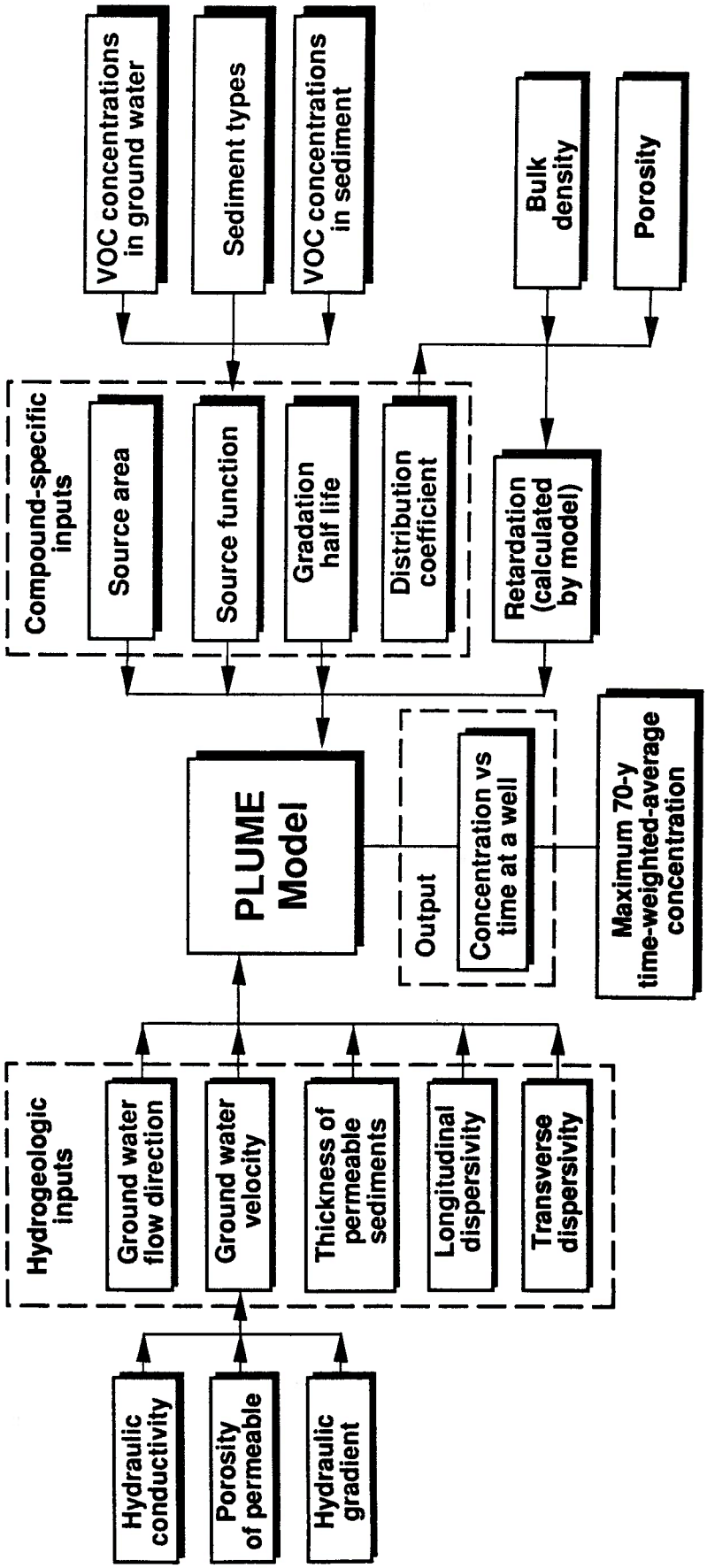

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Figure 4-6. Flowchart for PLUME modeling.

The main advantage of PLUME is the capability of simulating the migration of extensive, irregularly shaped plumes in a horizontal plane. As we will describe in more detail, this is accomplished by specifying rectangular source areas, each containing a uniform mass of contaminant. By superimposing many of these rectangles, the present distributions (concentrations) can be represented. However, each contaminant species must be simulated with a separate calculation. Several options are available for displaying the output, including areal plume configurations and concentration profiles along a line for future points in time, and concentration time-histories at selected locations in the domain.

The primary limitation of the model is the inability to simulate spatial and temporal variations in the media parameters that may produce variations in the direction and rate of ground water flow. Although a more complex numerical model could simulate such two-dimensional effects, it would likely result in longer travel paths and longer transport times. The PLUME code is an appropriate model for the present purpose, because we assume the shortest possible transport paths. It yields the most conservative 70-y concentrations for the compounds of concern (i.e., PCE, TCE, chloroform, and the "other VOCs") identified in Section 3 of this document, at all 12 observation points, which are used in Section 5 to estimate the baseline health risk.

4.3.2. Code Verification

To verify the numerics and accuracy of PLUME, we compared its output for one- and two-dimensional problems against analytical solutions available in the literature (Javandel *et al.*, 1984; Peaudecerf and Sauty, 1978). Three simulations were performed:

1. One-dimensional flow and dispersion in an aquifer where the contaminant is injected at a constant rate.
2. One-dimensional flow with an instantaneous point source and two-dimensional dispersion.
3. One-dimensional flow with a declining-strength point source with two-dimensional dispersion.

4.3.2.1. One-Dimensional Flow and Dispersion, Constant Source

For the fully one-dimensional problem, we modeled a homogeneous, isotropic aquifer of 10 m thickness and infinite extent. The seepage water velocity was constant at 1 m/d. An infinitely long ditch cut through this aquifer perpendicular to the direction of flow is shown in Figure 4-7. At time $t = 0$, a nonreactive contaminant is continuously released to the aquifer from the ditch at a rate of 0.1 m^3/d per unit length and at a concentration C_o = 10 kg/m^3. A longitudinal dispersivity of 10 m and porosity of 0.2 are assumed for the aquifer. The one-dimensional formulation is:

$$\partial D_L\left(\frac{\partial^2 C}{\partial x^2}\right) - F(v\partial C,\partial x) = \frac{\partial C}{\partial t}. \qquad (4\text{-}2)$$

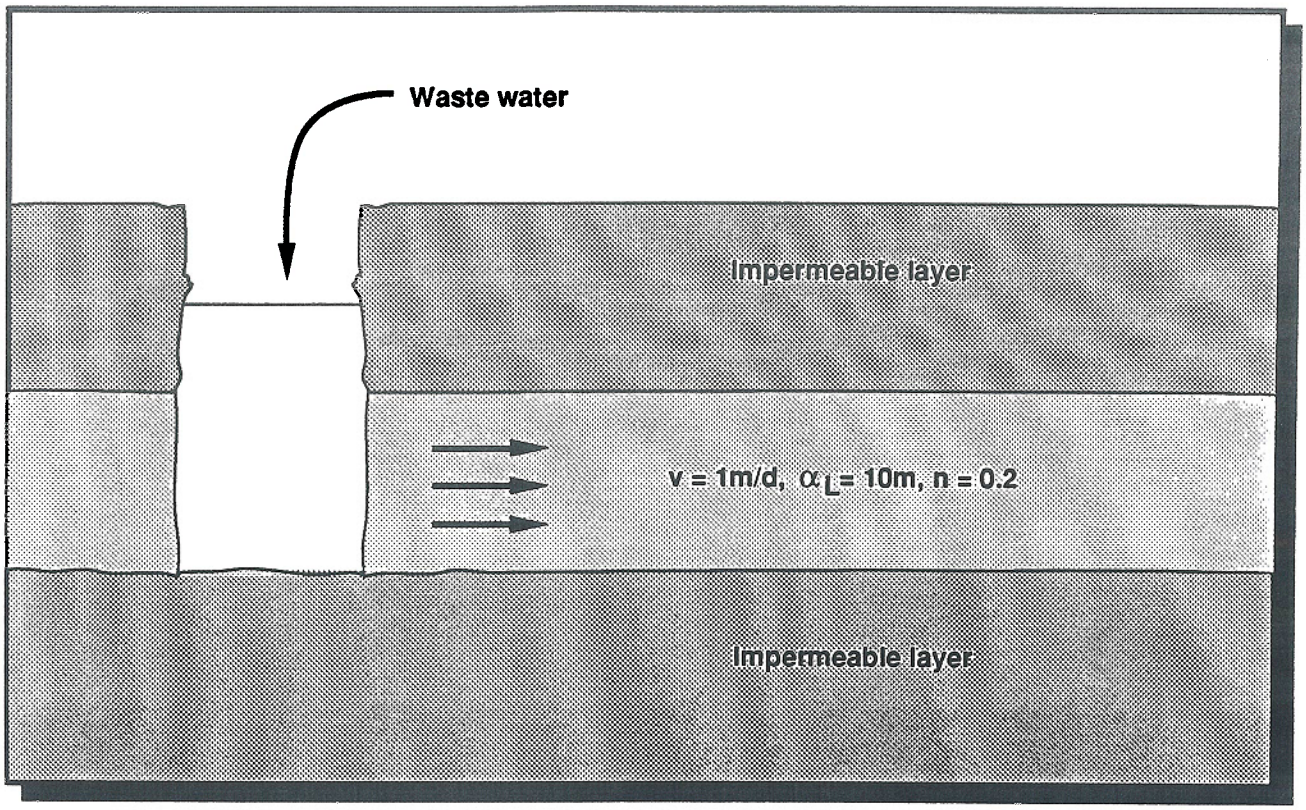

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Figure 4-7. Schematic diagram for one-dimensional verification problem.

The solution is (Javandel *et al.*, 1984):

$$\frac{C}{C_o} = A_3(x,t) \qquad 0 < t \leq t_o\,, \tag{4-3}$$

$$\frac{C}{C_o} = A_3(x,t) - A_3(x,t-t_o) \qquad t > t_o\,, \tag{4-4}$$

where

$$A_3(x,t) = \frac{1}{2}\,erfc\left[\frac{x-vt}{2(D_L t)^{1/2}}\right] + \left(\frac{v^2 t}{\pi D_L}\right)^{1/2} \exp\left[\frac{-(x-vt)^2}{2(D_L t)}\right]$$

$$-\frac{1}{2}\left[1 + \frac{vx}{D_L} + \frac{v^2 t}{D_L}\right]\exp\left[\frac{vx}{D_L}\right] erfc\left[\frac{x+vt}{2(D_L t)^{1/2}}\right], \tag{4-5}$$

and

C = solute concentration at time t and at distance x (kg/m3),

Co = solute concentration at x = 0, for t > 0 (kg/m3),

DL = longitudinal dispersion coefficient (= aLv) (m2/d),

x = distance from source (m),

v = seepage velocity in x direction (m/d),

t = solute injection time period (d), and

aL = longitudinal dispersivity (m).

To simulate the infinite dimensions of the problem, we assumed the ditch is 2,000 m in length, 0.1 m wide, and the midpoint of the ditch is at $x = 0$. Other parameters for the simulation were C_0 = 10 kg/m^3/unit length and a recharge rate = 0.1 m^3/d, which produce a chemical recharge mass rate of 1 kg/d/unit length. Results of the PLUME calculations are compared with the continuous, analytically derived breakthrough curves (Javandel *et al.*, 1984) in Figure 4-8. The close agreement verifies the one-dimensional solution in the code.

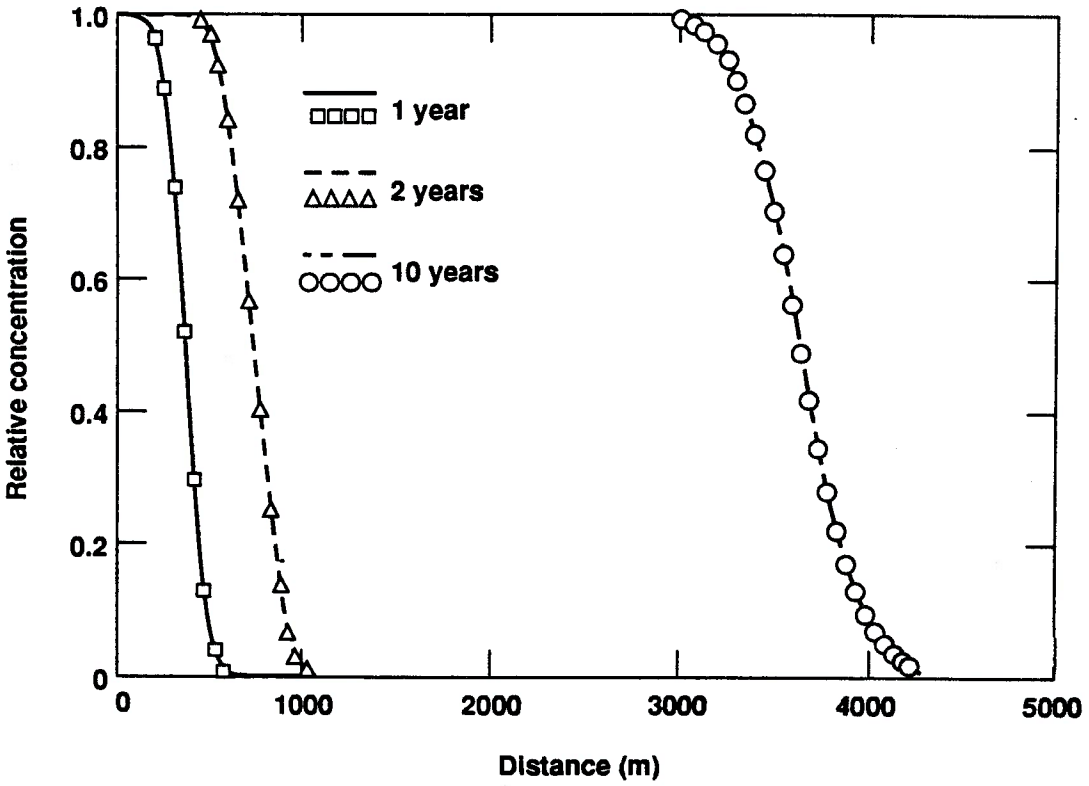

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Figure 4-8. One-dimensional analytical solutions and PLUME results showing concentration with distance from a one-dimensional, instantaneous point source at three observation times. (Lines represent analytical solutions and symbols show PLUME results.)

4.3.2.2. *One-Dimensional Flow with Two-Dimensional Dispersion, Instantaneous Point Source*

The partial differential equation describing two-dimensional solute transport is (Javandel *et al.*, 1984):

$$D_L \frac{\partial^2 C}{\partial x^2} + D_T \frac{\partial^2 C}{\partial y^2} - v\frac{\partial C}{\partial x} = R\frac{\partial C}{\partial t}, \tag{4-6}$$

where D_T is the transverse dispersion coefficient (= $\alpha_T v$) (m^2/d), α_T is the transverse dispersivity (m), and R is the dimensionless retardation factor.

For the second problem, Eq. (4-6) is solved for an instantaneous injection of solute of mass M_0 at the origin. The solution for this case is (Peaudecerf and Sauty, 1978):

$$C(x, y, t) = M_0 \frac{R}{4\pi b n D_L D_T}\left(\frac{1}{t}\right)\exp\left[\frac{-(x - vt/R)^2}{4D_L t/R} - \frac{y^2}{4D_T t/R}\right], \tag{4-7}$$

where n is the porosity (dimensionless), and b is the aquifer thickness (m). The following input values were used in the PLUME simulation:

M_0 = 100 kg,

n = 0.20,

b = 1 m,

v = 0.1 m/d,

α_L = 10 m,

α_T = 1 m, and

R = 1.0.

Analytical solutions are represented in Figures 4-9 and 4-10 as continuous and dashed lines. Figure 4-9 shows the concentration changes along the x-axis at y = 0 m and at y = 10 m, one year after the instantaneous solute injection of 100 mg. The maximum concentration has migrated about 36.5 m downgradient from the source. Figure 4-10 shows the concentration changes at two points (origin and x = 50 m, y = 5 m) at different times. The concentration decreases continuously with time at the origin, as expected. At the second point, the concentration increases up to about one year and declines thereafter.

For the PLUME simulation of this problem, a point source was idealized as a square with 0.1 m sides. The calculated concentrations along two lines (y = 0 m and y = 10 m) are plotted in Figure 4-9 by symbols. As shown in this figure, the match between PLUME output and analytical solution is excellent. The solute concentrations at various times calculated at the origin and at x = 50, y = 5 are shown in Figure 4-10 by symbols. Again, the match for the instantaneous point source is excellent.

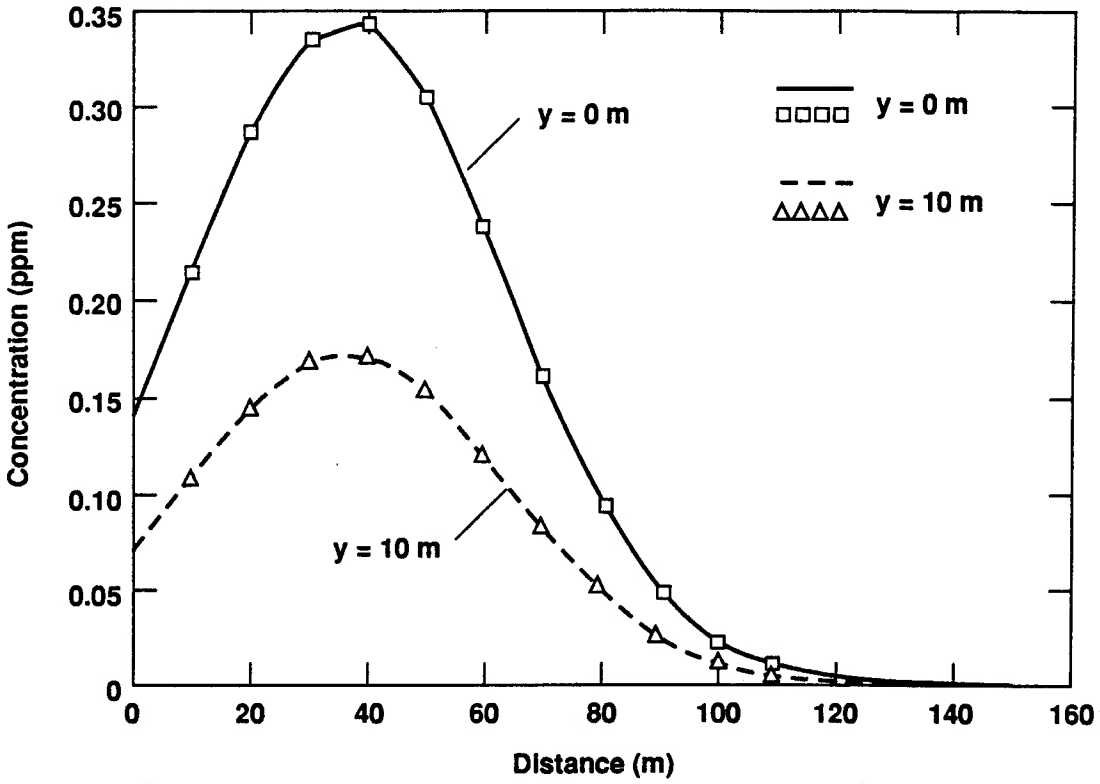

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Figure 4-9. Two-dimensional analytical solutions and PLUME results showing concentration with distance from a two-dimensional, instantaneous point source at an observation time of one year. (Lines represent analytical solutions and symbols show PLUME results.)

4.3.2.3. One-Dimensional Flow with Two-Dimensional Dispersion, Decaying Point Source

The third problem is similar to the previous, except that the solute mass has an exponentially declining release rate, defined by the source function:

$$dM/dt = M_0 e^{-kt}, \tag{4-8}$$

where

M_0 = total mass released,

k = $0.693/t_{1/2}$, and

$t_{1/2}$ = source strength half-life.

The solution to Eq. (4-6) for this case is (Bumb, 1989):

$$C(x, y, t) = \frac{1}{4\pi bn\,(D_L D_T)^{0.5}} \int_0^t \left(\frac{M_0 e^{-kt}}{t}\right) \exp\left[\frac{-(x - vt/R)^2}{4D_L t/R} - \frac{y^2}{4D_T t/R}\right] dt\,. \tag{4-9}$$

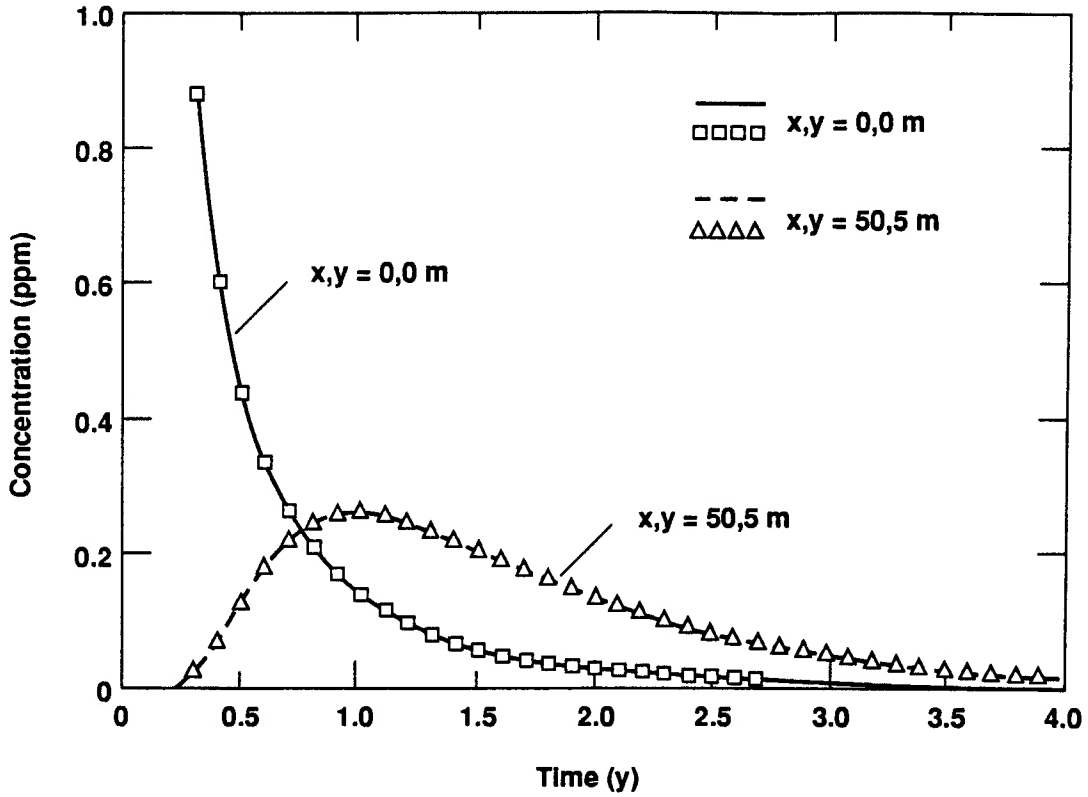

Figure 4-10. Two-dimensional analytical solutions and PLUME results showing concentration versus time for a two-dimensional, instantaneous point source at two observation points. (Lines represent analytical solutions and symbols show PLUME results.)

The solution is an integral summation of the solution for instantaneous point source [Eq. (4-7)], with the constant-mass term replaced by the variable-mass term of Eq. (4-8). An approximation of Eq. (4-9) was created by superimposing a series of 34 instantaneous point-source solutions, each with a mass release specified by Eq. (4-8). The mass inputs and timing of the set of instantaneous sources are shown in Figure 4-11. The result of this calculation was the concentration time-history for a point 500 m directly downgradient of the source, shown as the solid line in Figure 4-12.

In the direct PLUME simulation of the decaying source function, input parameters were the following:

$M_0 = 10$ kg,

$t_{1/2} = 10$ y,

$n = 0.30$,

$b = 1$ m,

$v = 0.1$ m/d,

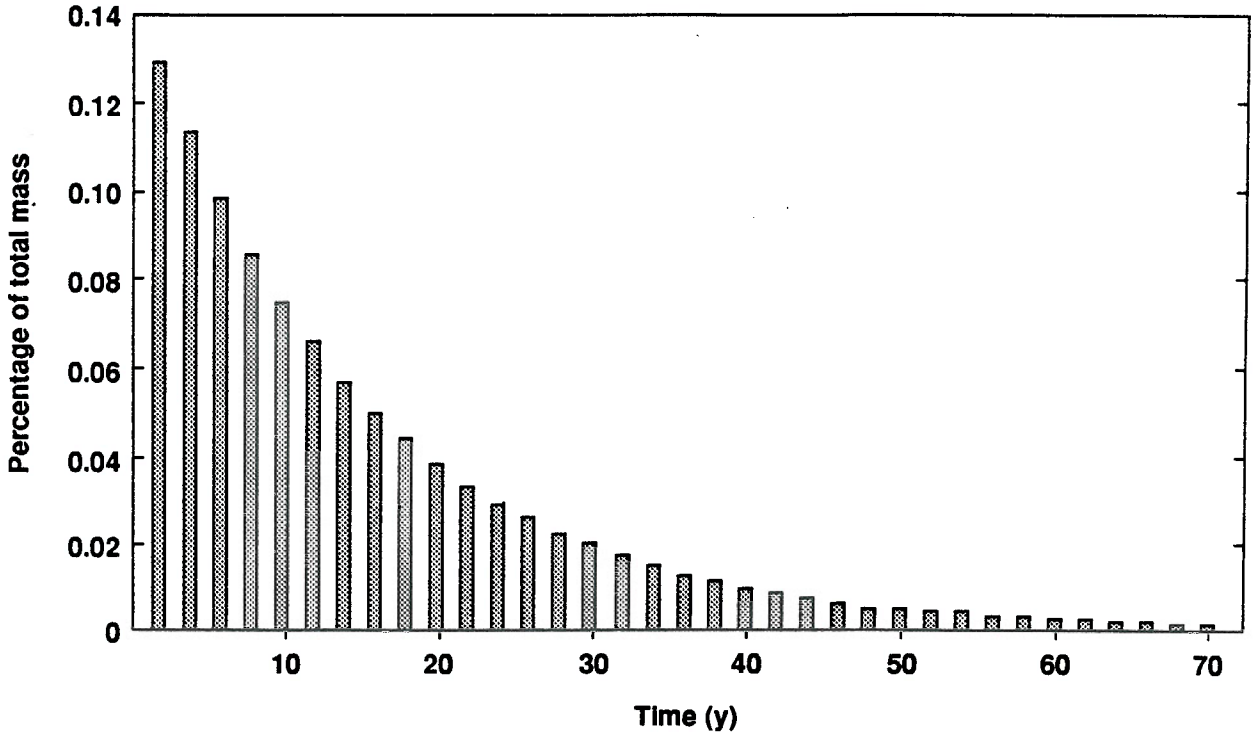

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Figure 4-11. Representation of a two-dimensional, exponentially declining point source by a series of instantaneous point sources.

α_L = 10 m,

α_T = 1 m, and

R = 1.0.

The point source was represented as a square with 0.1-m-long sides. The concentration predictions for a point 500 m directly downgradient are shown as small squares in Figure 4-12. The correspondence with the analytical solution is very good, with the exception of one anomalous point at 12 y, when conditions may be near a point where the code makes a transition between two solution algorithms. As discussed below, we use this decaying source option in the PLUME model to simulate leaching of remnant contaminants from the vadose zone and/or slow desorption of contaminants from fine-grained sediments in the saturated zone.

4.3.3. Parameter Sensitivity Studies

Before conducting migration calculations for the baseline risk assessment, we estimated the sensitivity of predicted contaminant concentrations in migrating plumes to variations of ground water flow velocity, dispersion, and degradation (Small *et al.*, 1988). For each test case, one parameter was varied over the estimated range of possible values, while keeping all other parameters constant.

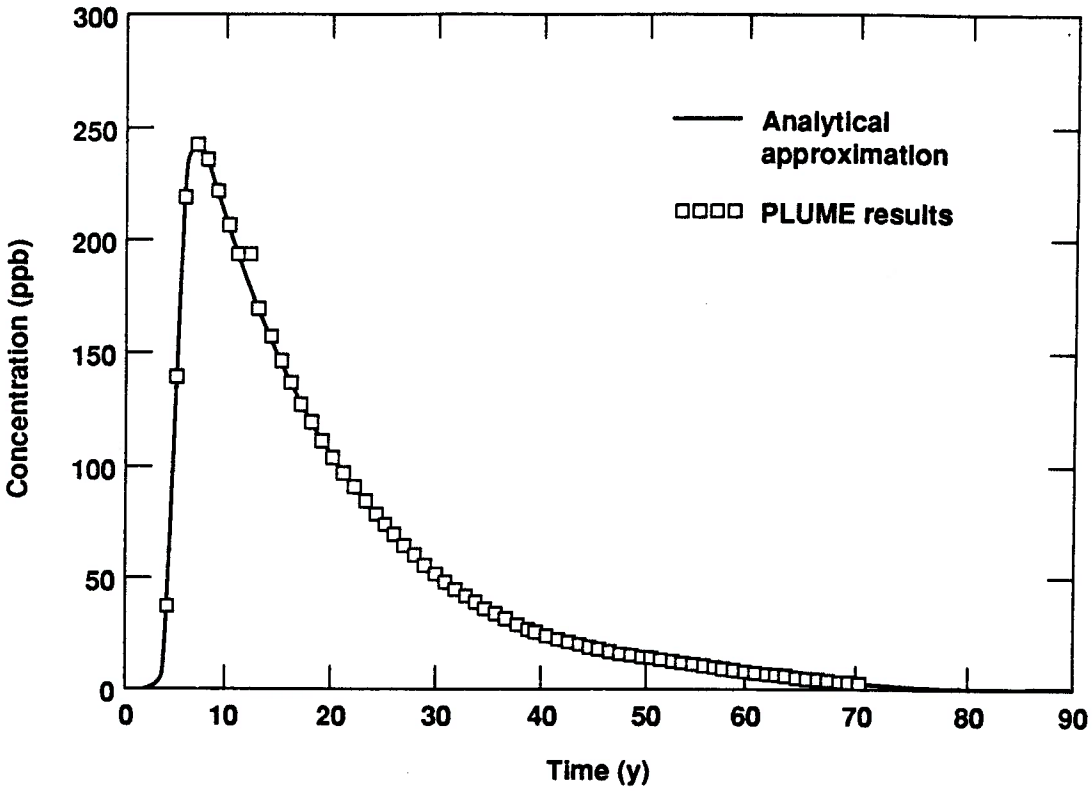

Figure 4-12. Concentration versus time for an analytical approximation and for PLUME results for a point 500 m downgradient of a two-dimensional, exponentially declining source.

These analyses showed that the model results are sensitive to variations in ground water velocity, which can affect the peak concentrations as well as arrival times. The model is less sensitive to variations in dispersion coefficients within the range considered here. Increasing dispersion coefficients produce similar concentrations, but at later times. A mid-range dispersion estimate yields a conservative combination of short arrival time, higher peak concentration, and moderate duration of exposure at a potential receptor well. The degradation half-life has the greatest effect on peak concentrations, especially at later times, with longer half-lives yielding higher concentration predictions. These findings have guided our choice of parameters for the two scenarios, as described later in this section.

4.3.4. Migration of Contaminants from the Vadose Zone to Ground Water

A preliminary qualitative estimate of migration and absorption in the vadose zone has been made for VOCs and the metals lead (Pb) and chromium (Cr). Vadose zone cleanup levels were then estimated by the Total Designated Level (TDL) methodology described by Marshack (1989). In the TDL method, an environmental attenuation factor (EAF) is applied to a water concentration (assigned for ground water protection) to determine the TDL value, according to the following equation:

$$\text{TDL} = \text{Water Quality Goal} \times \text{EAF} \qquad (4\text{-}10)$$

where the TDL represents the concentration of the soluble constituent in sediment waste (mg/kg) that could degrade water quality, the Water Quality Goal is the MCL (mg/L), and the EAF is a number based on the specific site conditions. A generic 100-fold attenuation factor has been used by EPA in the development of the EP TOX test, and by State of California DHS in their Waste Extraction Test (WET) test. Marshack (1989) provides qualitative guidelines for the development of EAFs for the migration of contaminants through the vadose zone to the ground water. He states that a generic 100-fold EAF can be used in situations which provide an "average" degree of natural protection for water quality from the discharge of waste under reasonable worst-case situations. An example of an average disposal situation is given as a landfill in the alluvium of the Central Valley where the depth to ground water is greater than 9 m and appreciable clay or silty-clay strata comprise the vadose zone.

The depth to ground water varies at LLNL from about 9 m in the northwest corner of the site to greater than 40 m in the southeast corner. In the potential source areas where vadose zone total VOC concentrations are greater than 500 ppb, the average depth to ground water is greater than 30 m. In areas where the ground water is about 15 m below the surface, VOC concentrations in the vadose zone are less than or equal to about 10 ppb. Similarly, lead and chromium have been found in ground water at LLNL in the areas where the depth to ground water is 30 and 15 m, respectively.

We have collected data to characterize the vadose zone at LLNL. Vadose zone cores have been analyzed for cation exchange capacity (CEC), particle distribution, and organic carbon content. CEC ranges from 1 to 30 meq/100 g. The percentage of clay-size particles varies from 1 to 43%. Organic carbon content is less than or equal to 0.1%. Vertical hydraulic conductivity and porosity of the vadose zone sediments are established at 10^{-7} cm/sec and 15 to 30%, respectively. Annual precipitation in the LLNL area is approximately 35 cm. Evapotranspiration in the area has been estimated to exceed the annual rainfall. The potential source areas where vadose zone total VOC concentrations exceed 500 ppb generally occur at facilities that are highly paved. To be conservative, however, no diminution in net recharge due to paving is assumed.

In the past, the total pollutant load has been sufficient to allow migration into ground water in excess of 1 ppm total VOC in the eastern portion of the site, and locally in excess of 10 ppm total aromatic hydrocarbons in the Gasoline Spill Area. Consequently, evidence supports the fact that, at least at some locations onsite, conditions existed to allow migration of contaminants through the vadose zone. Practices that led to significant releases are no longer operative at LLNL, and actual concentrations in the vadose zone are documented in Section 4 of the RI report (Thorpe *et al.*, 1990).

Lead and chromium are the metals of principal concern because they have been established as being present in LLNL ground water in concentrations exceeding the MCLs. Lead in the ground water underlying the site probably will occur as Pb^{+2}. In this form, it can be adsorbed onto negatively charged clay exchange sites, and is readily precipitated as an oxide. Pb^{+2} has a wide range of Eh (reduction-oxidation potential)–pH (acidity-alkalinity state) stability. At LLNL, lead is found in the subsurface associated with the Gasoline Spill Area. Organic compounds can enhance the mobility of metals in two ways. First, the consumption (or oxidation) of the solvent through biodegradation can cause reducing conditions, which can prevent precipitation of the

metal as an oxide. Second, the solvent can act as an organic ligand, which can increase the solubility of the metal (Stone and Morgan, 1984; Longmire, 1986).

The chromium species most prevalent in the ground water underlying the site is hexavalent chromium (Cr^{+6}). Most likely, chromium in this valence state is found in the ground water as the chromate oxyanion CrO_4^{-2}. This ion is stable in oxidizing/alkaline waters, down to a pH of about 6.4 (Hem, 1985; Dragun, 1988). Although Eh data are unavailable, wells containing Cr^{+6} generally have a pH of about 7.5. At lower pH, but still under oxidizing conditions, the Cr^{+6} would probably be in the oxyanion form of $HCrO_4^{1}$. The chromium oxyanions are generally considered anthropogenic ions, although highly alkaline/oxidizing waters can cause dissolution and oxidation of the Cr^{+3} from chromium oxides or hydroxides in minerals (Robertson, 1975). Such alkaline/oxidizing conditions may exist at LLNL.

From an anthropogenic source, the chromate oxyanions would tend to be more mobile through the subsurface than the metal cations. Even with sufficient clay mineral exchange sites available, anion exchange capacity is usually 5 to 15% of the CEC. The adsorption or distribution coefficients (K_ds) calculated for Cr^{+6} are about 1.5 to 2 orders of magnitude lower than K_ds for Cr^{+3} (Dragun, 1988).

Although lead, chromium, and VOCs may be fairly mobile in the LLNL vadose zone, the site conditions appear to be more protective of ground water contamination compared to the "average" site discussed earlier. The great depth to ground water and the extremely limited amount of recharge have the greatest impact on the attenuation capacity of the vadose zone. Further work is continuing to fully examine the effect of CEC and clay content on metal attenuation. As a conservative assumption, we estimate that EAFs for VOCs and metals are greater than 100.

The MCL for lead and chromium is 0.05 mg/L (see Table 3-5). Utilizing Eq. (4-10), a TDL for these metals in sediment would be greater than 5 mg/kg. For PCE and TCE, the MCLs are both 0.005 mg/L (see Table 3-5). Applying Eq. (4-10) demonstrates that the TDL should be greater than 0.5 mg/kg. We are conducting a more rigorous quantitative evaluation to support the cleanup levels that will be presented in the FS.

4.3.5. VOC Migration Calculations for Ground Water

4.3.5.1. Source Simulation

Input sources to PLUME were based on the 1988 isoconcentrations for PCE, TCE, chloroform, and "other VOCs," shown in Figures 4-13 through 4-16, respectively. Plumes for each of the four contaminants were approximated by the overlapping rectangles and corresponding masses shown in Figures 4-17 through 4-20.

The contaminant masses assigned to each rectangle were determined as follows. First, an isopach map showing the cumulative thickness of contaminated, permeable sediments was constructed. Areas where the isoconcentrations and isopachs intersected were measured and

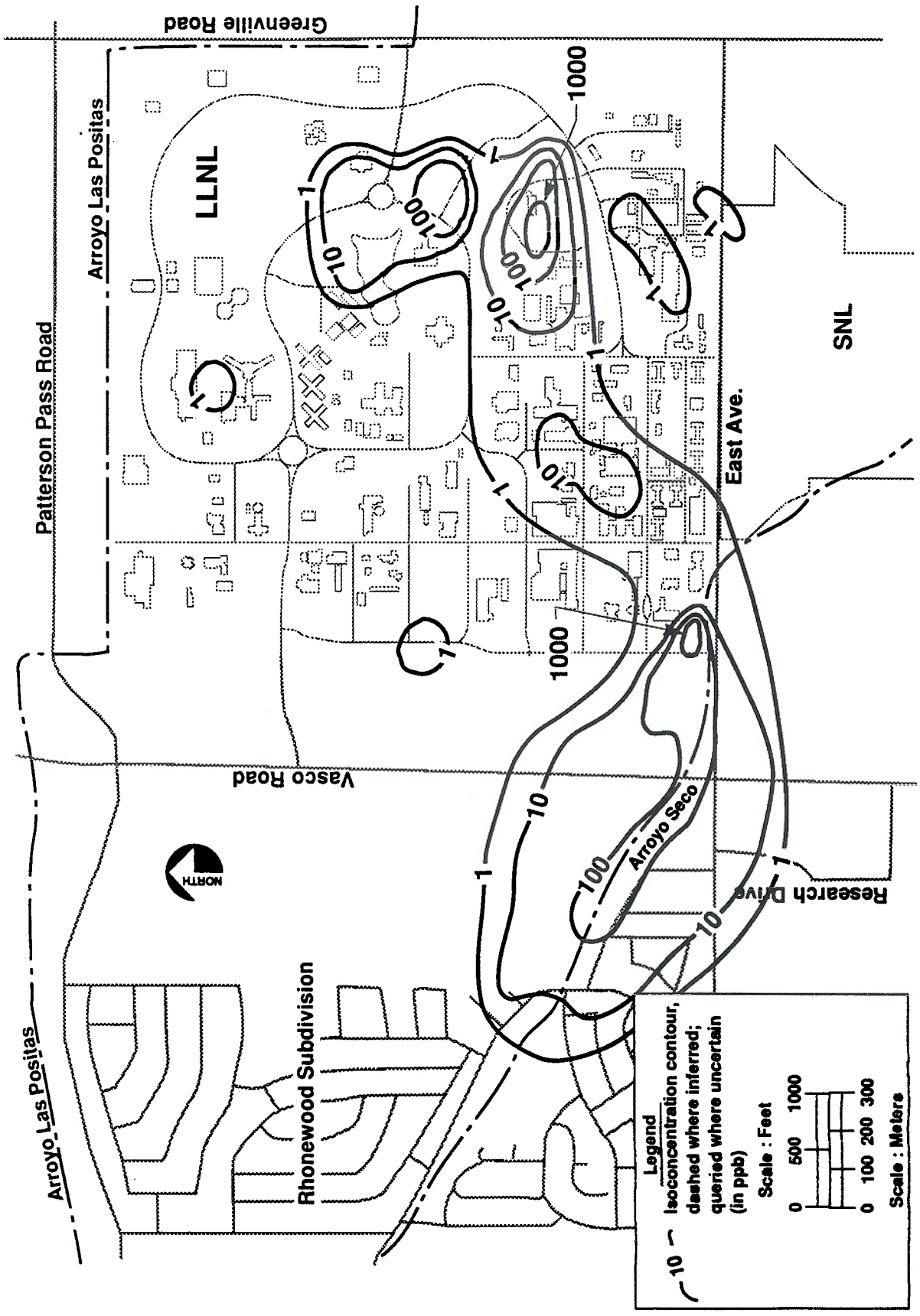

Figure 4-13. Isoconcentration contour map of PCE in ground water, LLNL and vicinity, January - September 1988.

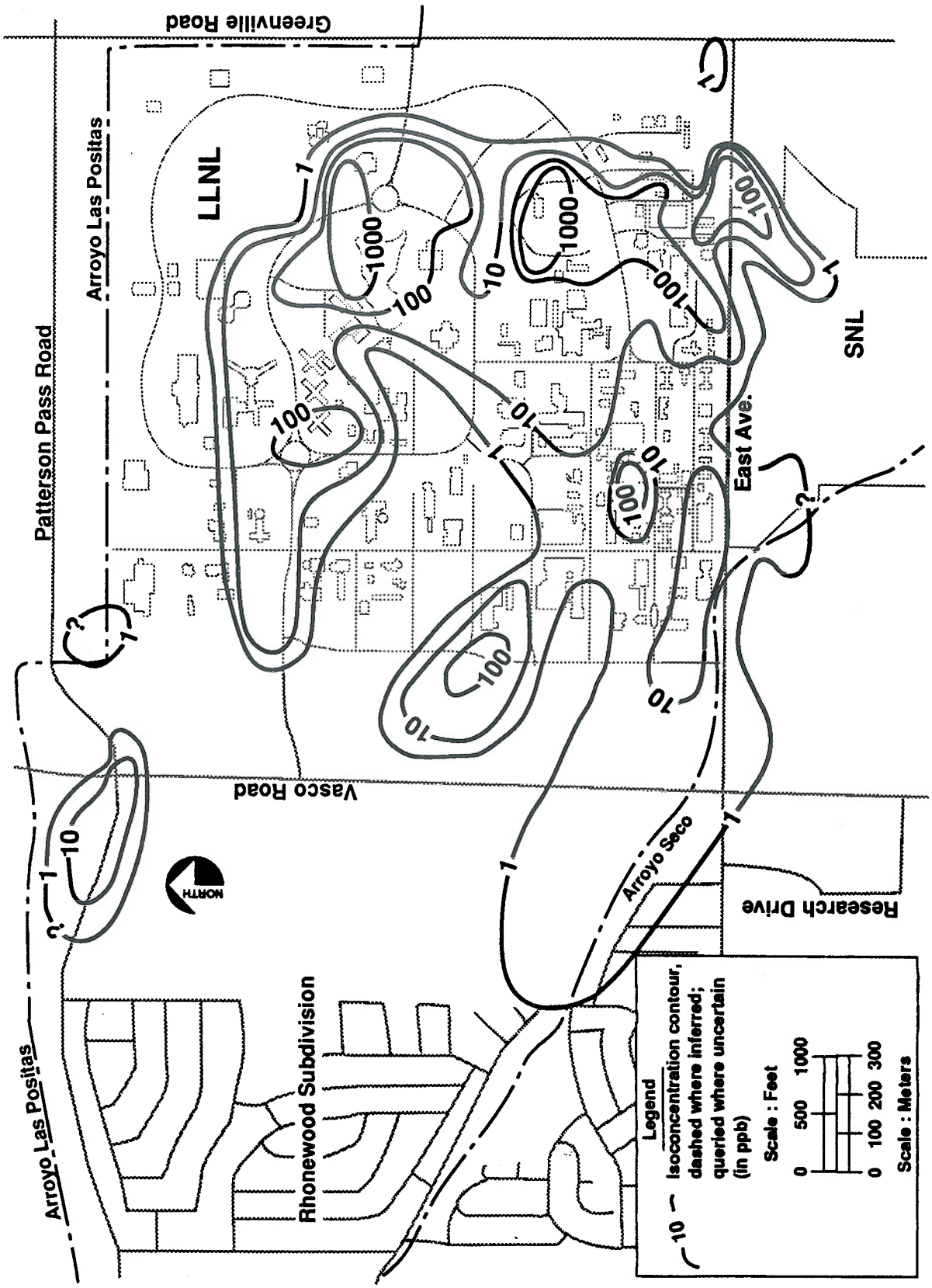

Figure 4-14. Isoconcentration contour map of TCE in ground water, LLNL and vicinity, January - September 1988.

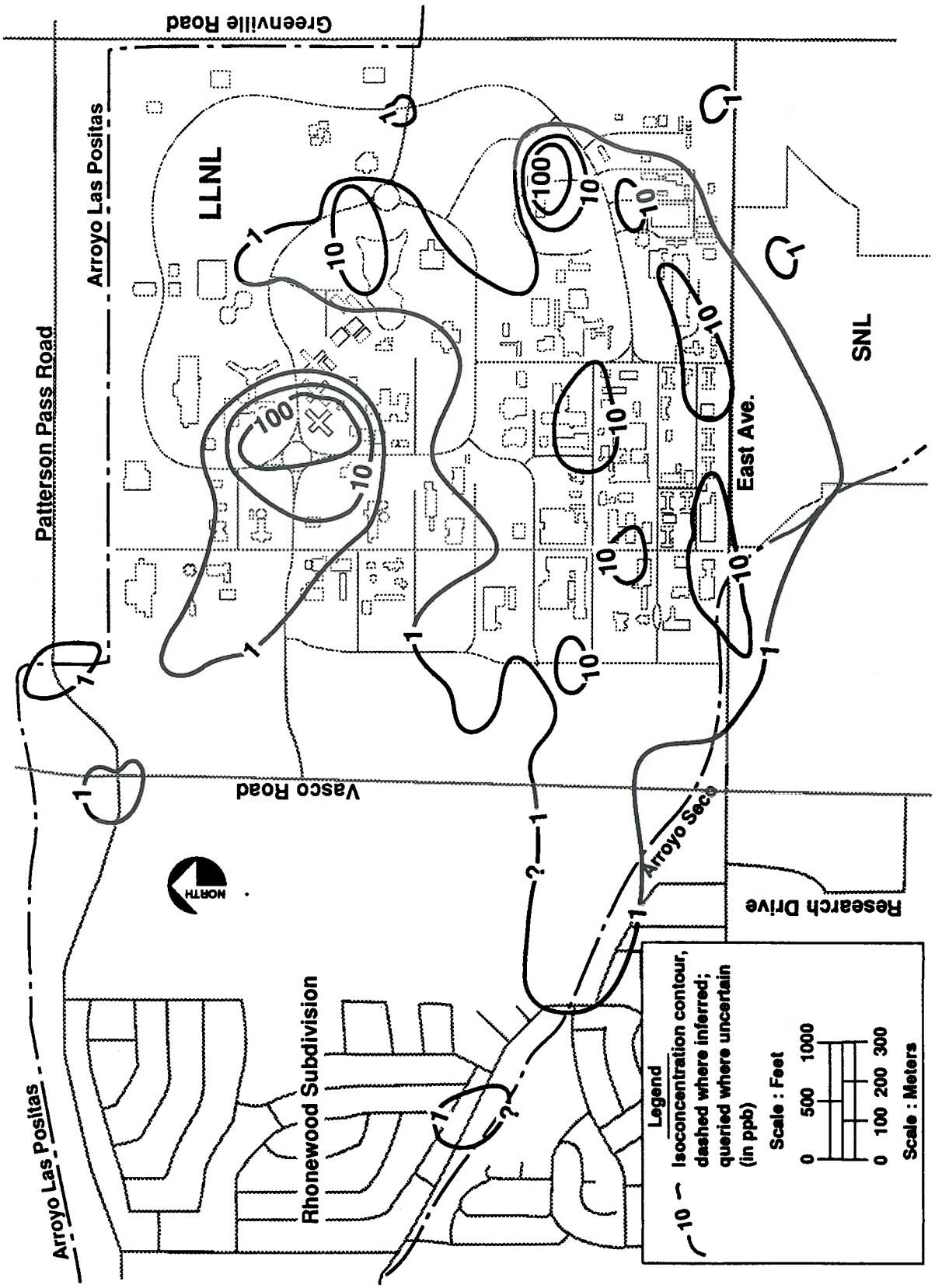

Figure 4-15. Isoconcentration contour map of chloroform in ground water, LLNL and vicinity, January - September 1988.

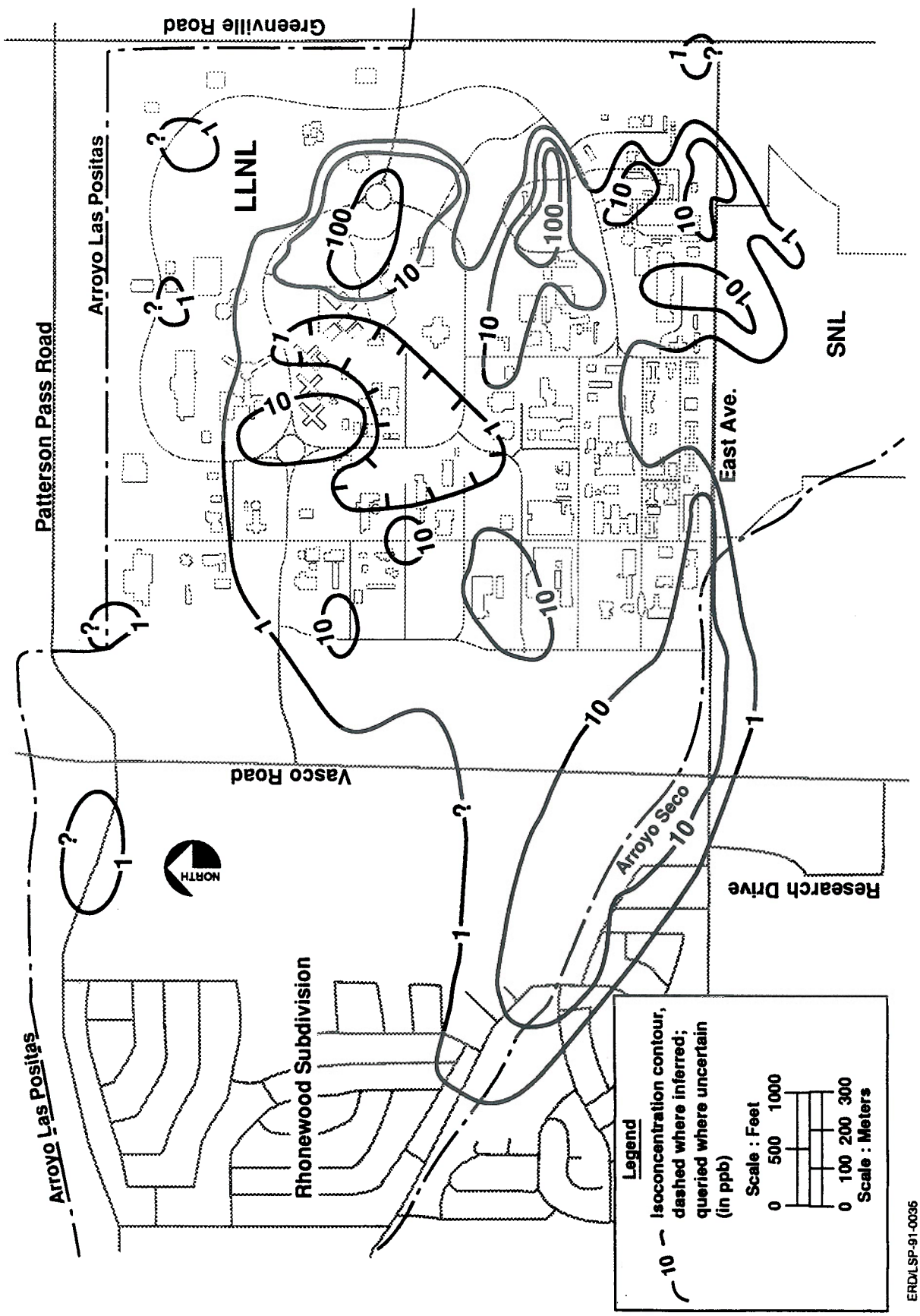

Figure 4-16. Isoconcentration contour map of "other VOCs" (excluding TCE, PCE, and chloroform) in ground water, LLNL and vicinity, January - September 1988.

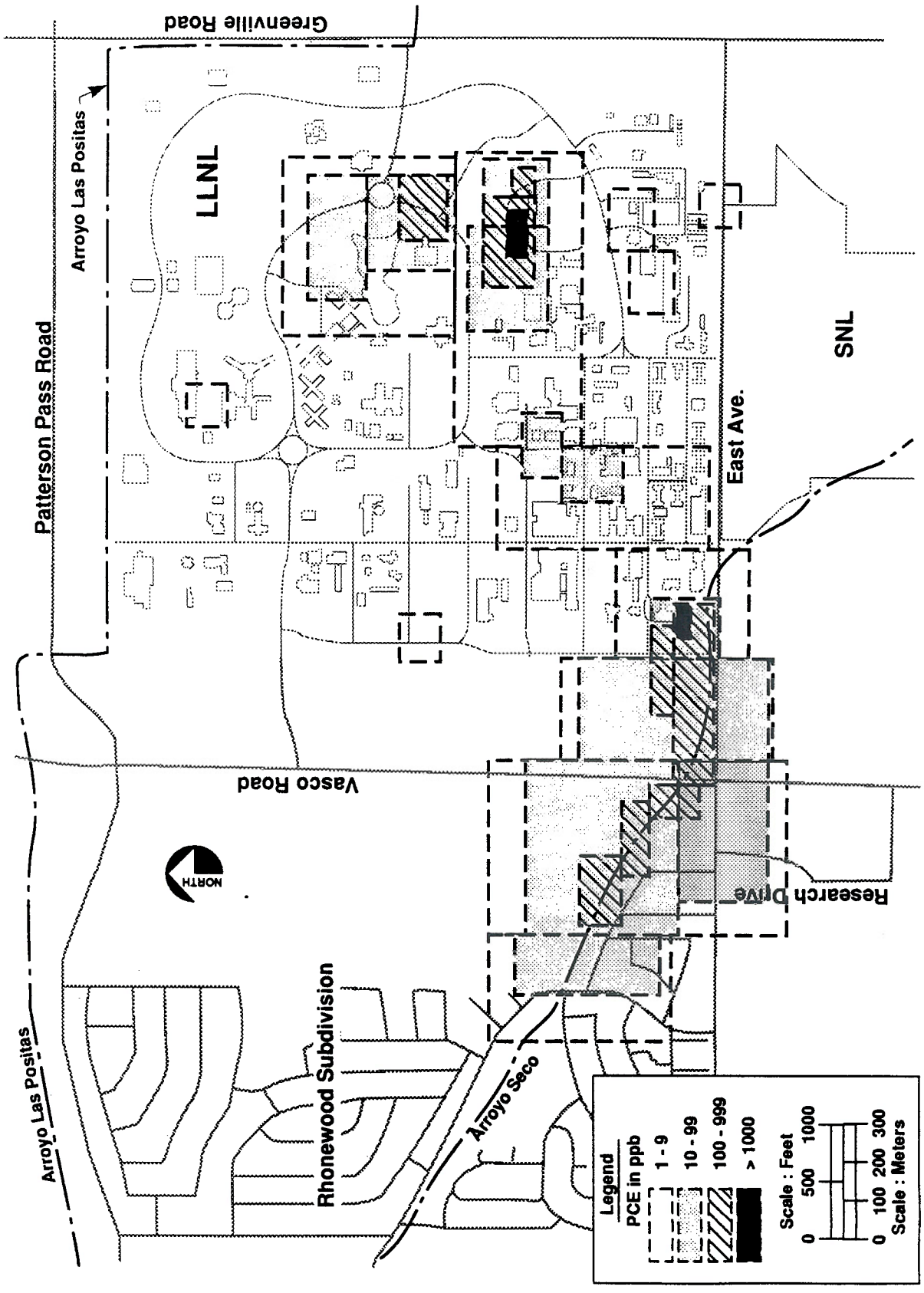

Figure 4-17. Input source rectangles for PCE for PLUME migration simulations.

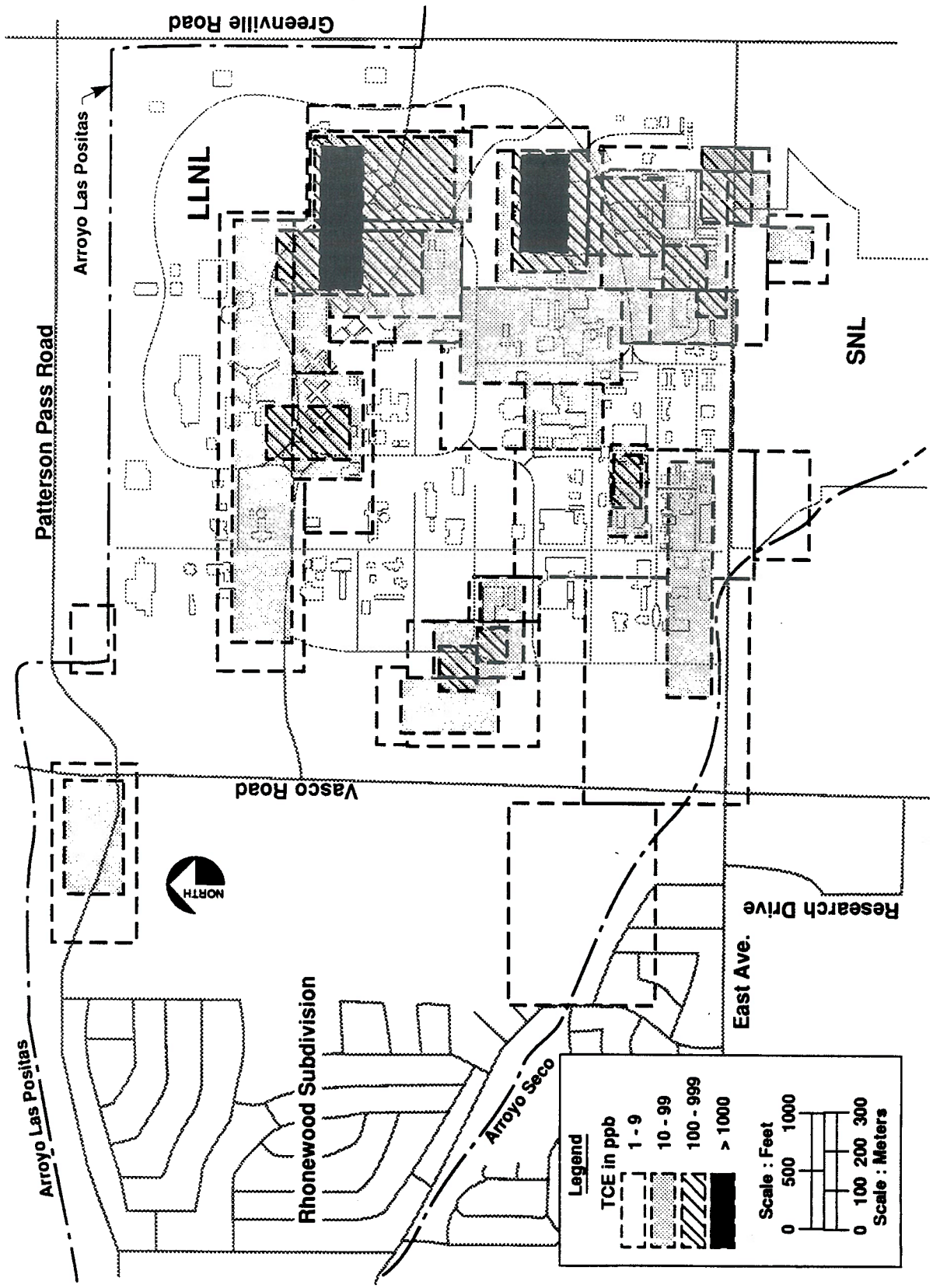

Figure 4-18. Input source rectangles for TCE for PLUME migration simulations.

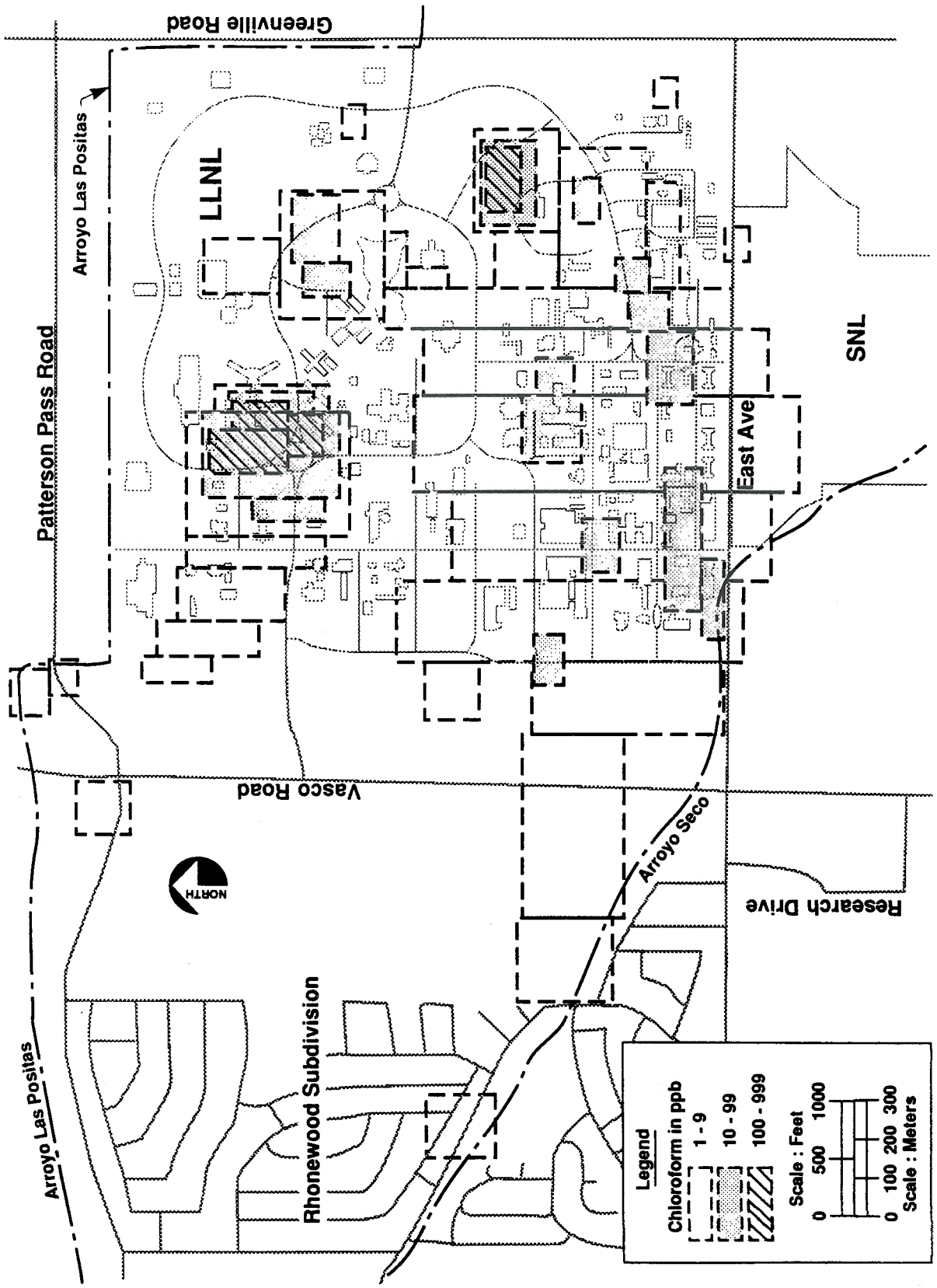

Figure 4-19. Input source rectangles for chloroform for PLUME migration simulations.

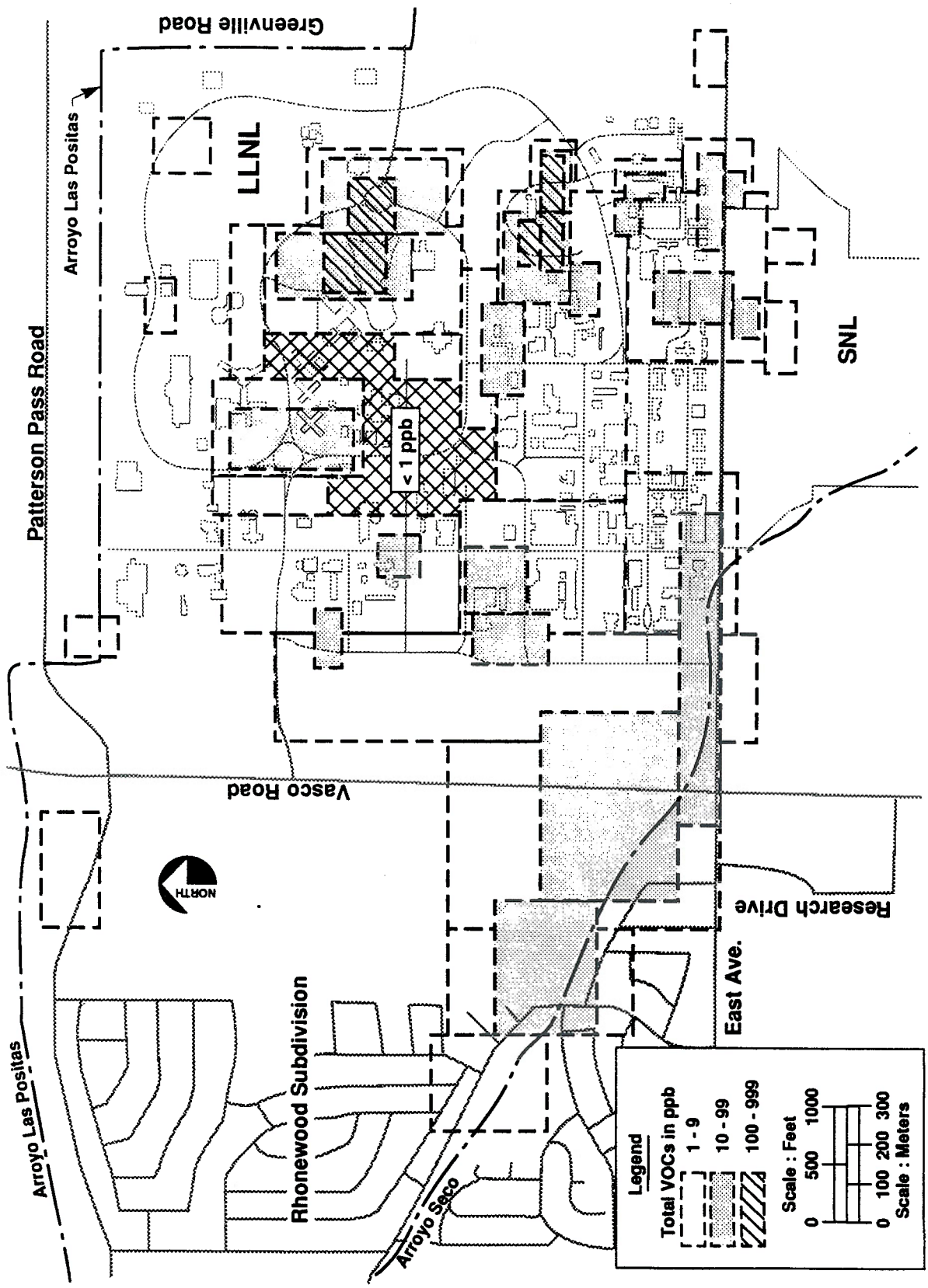

Figure 4-20. Input source rectangles for "other VOCs" (excluding TCE, PCE, and chloroform) in PLUME migration simulations.

multiplied by the average aquifer thickness over that area. The sum of these volumes was then divided by the area within the isoconcentration to yield an area-weighted average thickness. Finally, the contaminant mass for a given rectangle was calculated as the product of its area, the area-weighted thickness, an assumed porosity of 0.30, and the average concentration over that area. For example, a rectangle representing 10 to 99 ppb was assigned a concentration of 55 ppb. Additional source rectangles were superimposed to represent successively higher isoconcentration levels, and the mass of each overlying rectangle was adjusted to account for the contaminant mass already represented by underlying rectangles. As discussed below, these same rectangles were used to represent areas where additional contaminants might be released slowly over the future from both the vadose zone and from saturated, sorptive sediments.

We used an instantaneous source function to establish existing contaminant concentrations in ground water. Contaminants from this type of source will tend to migrate as a pulse, whose center of mass has a velocity equal to the mean seepage velocity divided by the retardation factor, R. The instantaneous source function is not sufficient to simulate conditions at the LLNL site because (1) additional contaminants are probably bound to fine-grained sediments in the saturated zone, and (2) some residual contaminants remaining in the vadose zone may have the potential to reach ground water. Slow desorption from saturated sediments and/or leaching from the vadose zone may be providing contaminants to the ground water on a delayed basis. The center of mass of the onsite plume is still within the LLNL-site boundary after about 40 y of migration, which suggests considerable retardation in the vadose zone and slower release to ground water.

We approximated the longer duration introduction of contaminants to ground water by superimposing an exponentially declining source function on the same rectangular sources used for the instantaneous release. For the best estimate, the amount of mass available for the slow-release source function was estimated to be an additional 50% of the mass released in the instantaneous simulations, and an additional 100% of the instantaneously released mass was used for the health-conservative estimate. Soil chemistry data from several known release sites indicate that these values are quite conservative. Available data indicate that there are no large masses of separate phase VOCs held within either fine-grained, saturated sediments or the unsaturated zone. The specified mass for the exponentially declining source function does not greatly affect peak concentrations predicted by PLUME. Rather, this parameter affects the duration of observable levels of contaminants predicted by the model.

The initial concentration of contaminants in ground water due to an instantaneous source is also affected by the retardation factor, R. The PLUME code assumes that a portion of the specified mass is initially sorbed, so that only $1/R$ times the mass remains dissolved. To produce the correct mass dissolved in ground water, therefore, we multiplied the masses calculated for the ground water by R. For the health-conservative simulations, this was of no consequence, since no retardation was assumed, and all the mass entered the ground water.

A history-matching study was conducted to determine how best to represent the delayed release of contaminants to the ground water. Previously, we demonstrated how the exponentially declining source half-life determines the rate at which an input mass is released to ground water.

An appropriate source half-life was determined by two calibration simulations in which all input parameters, except source half-life, were held constant and the half-life was varied. For the best-estimate scenario of 50% additional (delayed release) mass, it was found that a 10-y source

half-life was the shortest release rate that did not cause the predicted concentrations to exceed the value of present-day observed concentrations. Similarly, it was found that a 20-y half-life was appropriate for the health-conservative release rate for 100% additional mass. Thus, both the best-estimate and health-conservative release rates are consistent with the fact that 5 y of monitoring indicate that concentrations within the existing plumes generally have not risen at the LLNL site.

4.3.5.2. Estimation of Input Parameters

As described earlier, both best-estimate and health-conservative scenarios are considered to address uncertainties in the input parameters. These two scenarios represent different estimates for ground water flow velocity, dispersion, retardation, and degradation rate. Two different source masses of VOCs, including those dissolved in ground water, sorbed on fine-grained sediments, and suspended in the vadose zone are also used. The best-estimate parameters are values that we believe are the most likely or representative, based on current knowledge of the ground water system and VOC properties and distributions. The health-conservative parameters were chosen as the maximum plausible (upper bound) values for each parameter, based on existing statistical distributions. Although concentrations are calculated for the near- and mid-field wells, they are not considered in the risk calculation in Section 5 under the best-estimate case, because the scenario for them to exist is highly unlikely. However, we predict best-estimate concentrations at the far-field observation points for comparison with the results of the health-conservative scenario.

Input parameters used for both the best-estimate and health-conservative simulations are listed in Table 4-2 and discussed below. More detailed explanations of some of the technical considerations of parameter estimates are presented next and are discussed further in the cited references.

4.3.5.2.1. Porosity and Bulk Density. Values of porosity were calculated using measurements of bulk density (ρ_b) and specific grain density (ρ_s) for Livermore site sediments. The calculated porosities appear to be normally distributed (Fig. 4-21). The mean value of 0.30, which is typical of alluvial sediments (Freeze and Cherry, 1979), was used in the model to calculate pore volume and the retardation factor. Ground water velocity, which also is calculated using porosity, is explicitly entered into the model. The relatively small variation in porosity has a less significant effect on ground water velocity than the large variations in hydraulic conductivity. Therefore, we chose a single value of porosity (0.30) to estimate velocity, and used the variations in hydraulic conductivity to estimate the range of velocities for the best-estimate and health-conservative scenarios.

4.3.5.2.2. Thickness of Permeable Sediments and Contaminant Masses. PLUME requires a single, constant thickness of permeable sediments. To derive an estimate of this value from field data, we created an isopach map of contaminated sediments and calculated the area-weighted average thickness, as described above. This process was repeated for each compound modeled. Calculated sediment thicknesses for PCE, TCE, chloroform, and "other VOCs" are 4.2, 4.7, 6.0, and 3.7 m, respectively. Because the estimated thicknesses of permeable sediments were quite similar, we adopted a nominal value of 5 m for all of the VOCs

Table 4-2. Summary of PLUME input parameters for the best-estimate and health-conservative cases.

Parameter	Units	Best-estimate value	Health-conservative value
Hydrogeologic parameters			
Porosity (n)		30%	30%
Particle density (ρ_s)	g/cm^3	2.6	2.6
Thickness of saturated, permeable sediments containing VOCs (b)	m	5	5
Advection (v)	m/y	22	44
Dispersivity (α_L/α_T)	m	20/2.0	10/1.0
Compound specific parameters			
Distribution coefficient (K_d):			
PCE		0.42	0
TCE		0.20	0
Chloroform		0.083	0
"Other VOCs"[a]		0.16	0
Resulting retardation factor (R):			
PCE		3.8	1
TCE		2.2	1
Chloroform		1.5	1
"Other VOCs"[a]		2.0	1
Degradation half-life	y	50	No degradation
Percent additional available mass with respect to mass in solution		50%	100%
Release rate half-life of additional available mass	y	10	20

[a]See text for compounds included in this category.

in the simulations. However, the concentrations calculated at downgradient receptors are insensitive to this parameter, because we assume there is no change in the thickness between the source plumes and potential receptor wells.

The source masses for each compound presented in Table 3-13 in Section 3 of this document were calculated from the estimated thicknesses, porosity, and concentrations.

4.3.5.2.3. Ground Water Velocity. The PLUME model assumes a vertically averaged solute concentration and constant ground water velocity. At the LLNL site, sediments are horizontally stratified and thus characterized by a locally heterogeneous distribution in hydraulic conductivity and other material properties. This heterogeneity gives rise to local variations in

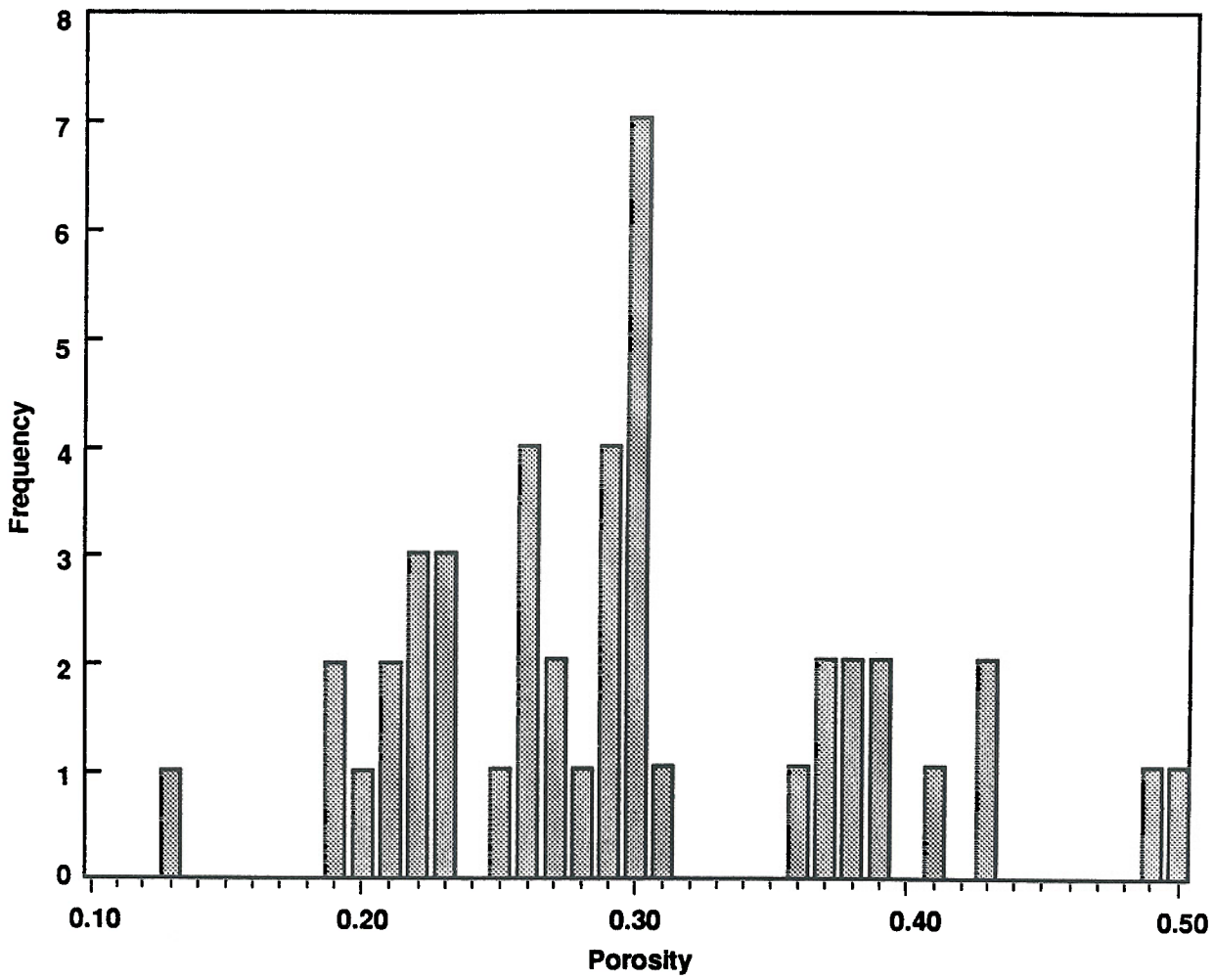

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Figure 4-21. Frequency distribution of porosity values derived from laboratory analysis of permeable sediments at LLNL.

ground water velocity and solute concentrations over scales of a few meters. However, over a larger observation scale of tens or hundreds of meters, this variation becomes less noticeable, and a uniform seepage velocity (advection term) can be used to simulate contaminant migration.

History-matching calibrations that utilize the contaminant as a tracer can be used to determine an appropriate uniform velocity over a large scale, if the source term is known. However, when precise information about actual releases is lacking, which is the case for the multiple releases at the LLNL site, the velocity can be estimated on the basis of statistical data on hydraulic conductivity and gradients. This process is currently the subject of much research and debate in the ground water profession, some of which we reference in the following discussion. In estimating velocity, we have assumed that a shorter transport time, resulting from higher estimated velocity, is more health-conservative. In other words, the sooner contaminants reach a receptor, the greater will be the health risk. We have also used approximate history-matching to confirm that these calculated velocities are reasonable.

Ground water seepage velocity, v, is calculated from the Darcy flow relation

$$v = -K_{\text{eff}}\, i / n\,, \tag{4-11}$$

where

K_{eff} = effective hydraulic conductivity,

i = hydraulic gradient (negative in the flow direction), and

n = porosity.

As described above, the porosity value used in the PLUME simulations is 0.30. Estimates of the current gradient made from head measurements across the study area, shown in Figure 4-22, are on the order of 0.004 beneath the LLNL site, 0.002 just to the west of the LLNL site, and 0.004 to 0.008 near the flow boundary between the Mocho I and Mocho II subbasins near downtown Livermore. In our judgment, the most representative value of i over the entire transport pathway is 0.004.

Hydraulic conductivity is highly variable beneath the site, as described in Section 3 of the RI report (Thorpe *et al.,* 1990). Results of pump tests in 167 wells in the area of the LLNL site show an approximate lognormal distribution (Fig. 4-23) in hydraulic conductivity. The geometric mean of this distribution is approximately 1.2 m/d or 30 gpd/ft^2. Recognizing that this mean value may not have any true physical significance, particularly over a large scale, Tompson (1990) performed an independent analysis of the data, using recently developed stochastic theories, to determine a more representative value. For the LLNL site and vicinity, he recommended that the effective conductivity be taken as 4.6 m/d, which is nearly four times the geometric mean.

Given these parameters, the best-estimate seepage velocity is 22 m/y. To place an upper bound on velocity, Tompson (1990) suggested that the hydraulic conductivity be doubled to 9 m/d. This yields a seepage velocity of 44 m/y, which we adopt as the health-conservative value for migration calculations.

4.3.5.2.4. Dispersion. Research indicates that the value of dispersivity is not a constant but increases with the distance traveled (Pickens and Grisak, 1981; Gelhar, 1987). This is known as "scale-dependent" dispersion. Dispersivities calculated from large-scale field experiments can be several orders of magnitude larger than those calculated from laboratory- or small-scale field experiments. This scale-dependent effect occurs because as the plume migrates it encounters larger-scale velocity variations, induced by the presence of geologic features such as lenses and layers. The variabilities tend to mix a plume over larger and larger scales. Gelhar and Axness (1983) predicted that if the variations in hydraulic conductivity, which give rise to the velocity variations, are statistically stationary, then this growth in dispersivity should tend toward a large but constant value, known as the asymptotic macrodispersivity. It is reasonable to choose dispersivities that are representative of the average scale of interest, such as the average distance over which the plume has traveled or will travel. Because the source strengths, locations, and durations of releases are not presently well known at the LLNL site, we cannot use existing data to calculate dispersivity. Instead, we will estimate dispersivity by citing experiments performed in similar geologic environments.

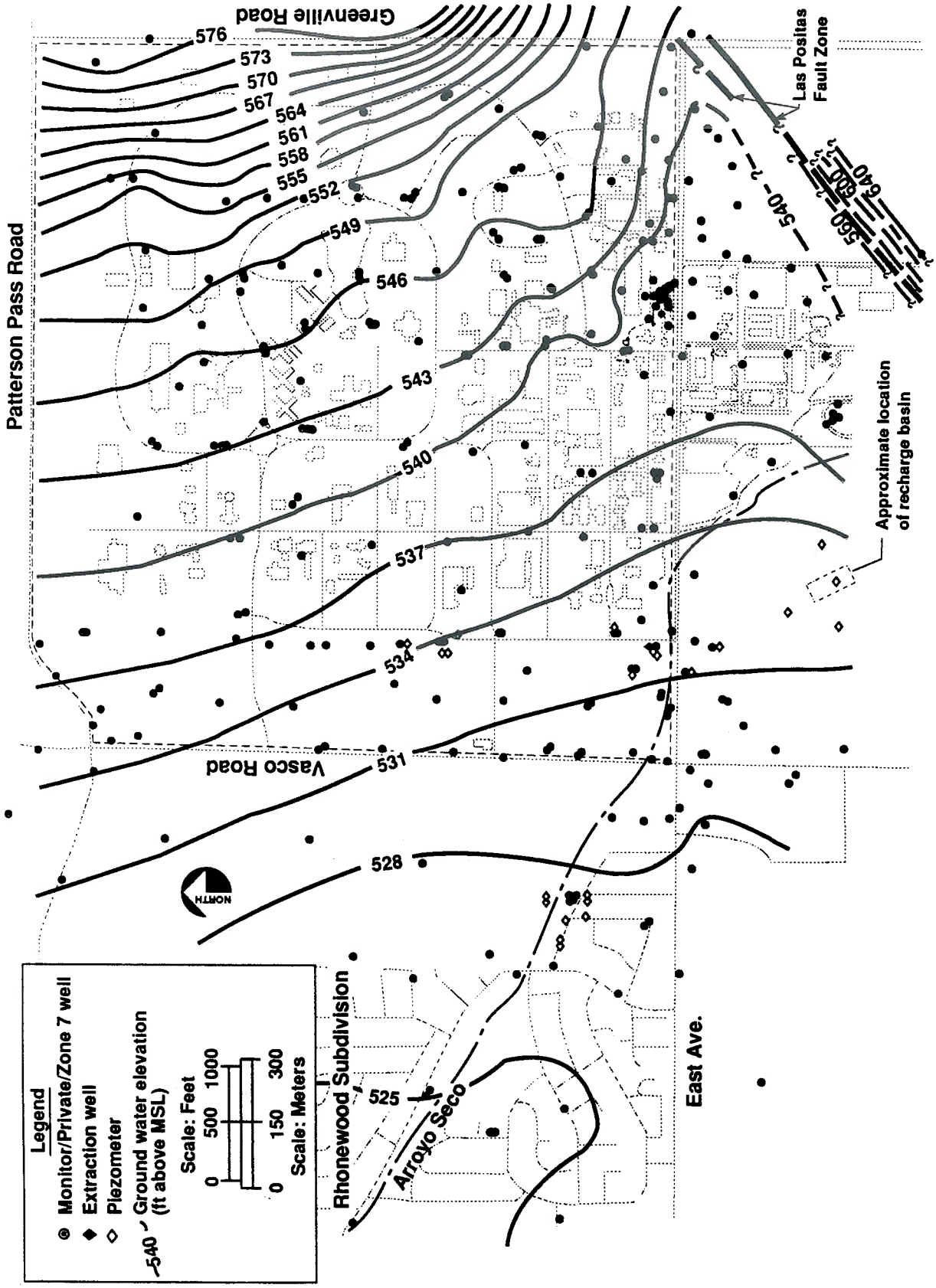

Figure 4-22. Ground water elevation contour map, March 1989, LLNL and vicinity.

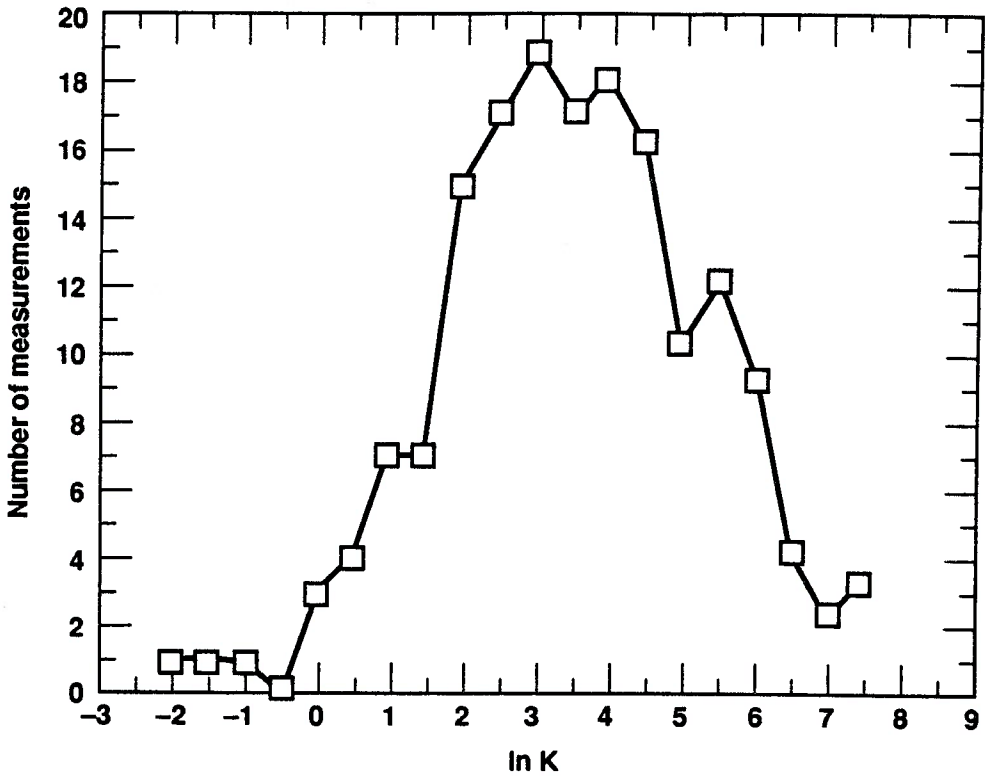

Figure 4-23. Frequency distribution of natural logarithm of hydraulic conductivity, K (ln gpd/ft^2) derived from hydraulic tests at LLNL.

There is a growing body of experimental work in which dispersivities have been measured in the field using tracer tests. Gelhar *et al.* (1985) presented a review of field-scale dispersivity measurements. The results are summarized in Figure 4-24. The shaded portion has been added to show the scale of this investigation, and it suggests a longitudinal dispersivity range of 3 to 300 m. However, the review includes experiments conducted with widely varying methods and in diverse geologic settings. Of the 99 values of longitudinal dispersivity cited and shown in the figure, seven represent field tests conducted in alluvium at scales greater than 100 m. These seven tests reported longitudinal dispersivity values of 12, 15, 20, 30.5, 41, 61, and 61 m.

Large dispersivities lead to faster contaminant migration and initial detection at downgradient receptors, but lower overall concentrations and accompanying lifetime exposures. For this reason, we have used values in the lower range of the experimental data. As listed in Table 4-2, the best-estimate longitudinal dispersivity (α_L) was chosen as 20 m. For the health-conservative case we used half this value (10 m), which is less than any of the experimental data.

Transverse dispersivity is typically at least an order of magnitude less than longitudinal dispersivity (Freeze and Cherry, 1979). The review by Gelhar *et al.* (1985) indicated that transverse dispersivity varies from essentially nil to less than 10 m in most geologic settings. Therefore, we assumed a transverse dispersivity equal to one-tenth the value of the longitudinal dispersivity, in both the best-estimate and health-conservative cases.

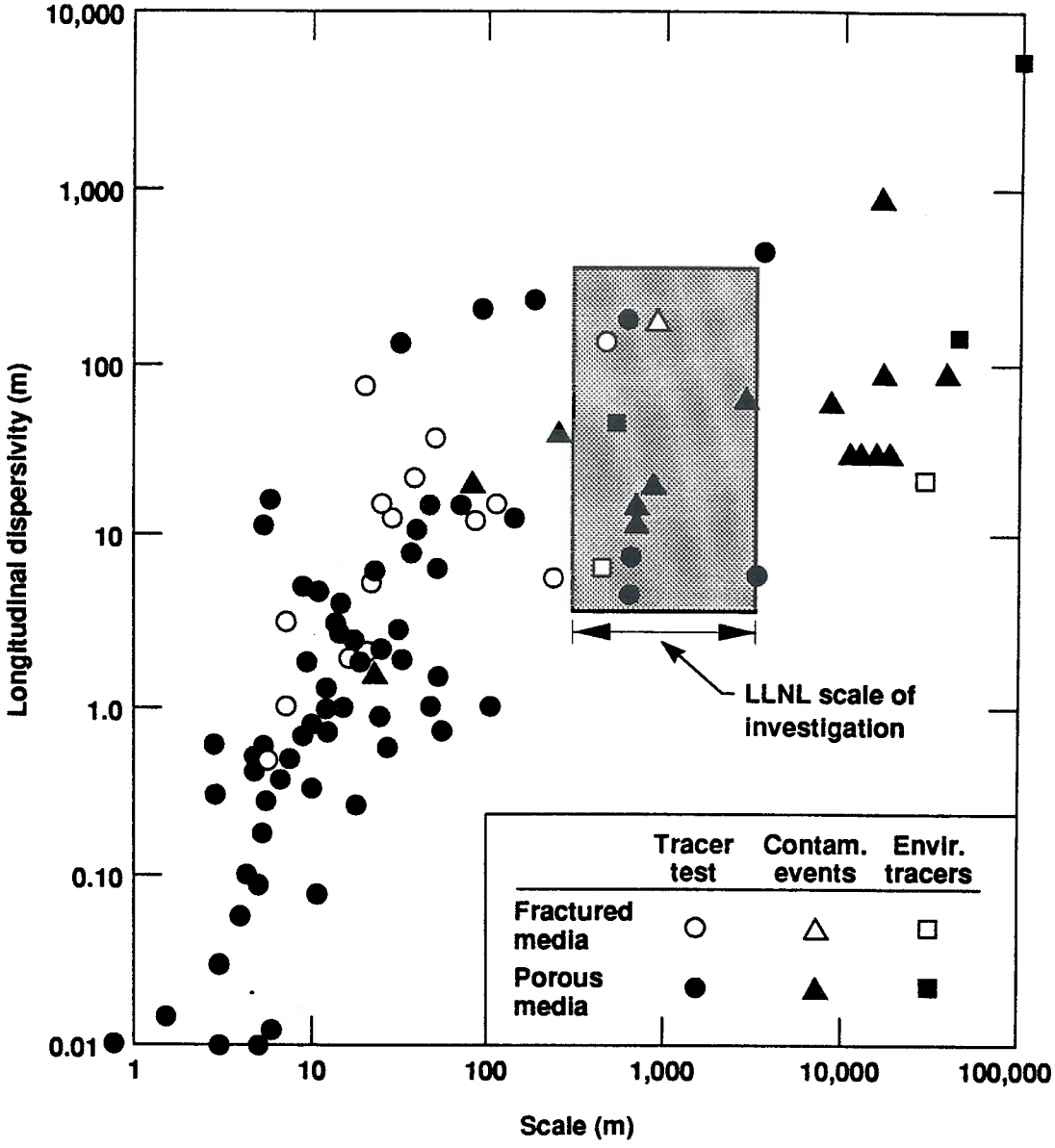

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Figure 4-24. Scale of observation versus longitudinal dispersivity for the saturated zone (adapted from Gelhar *et al.*, 1985).

4.3.5.2.5. Retardation. Retardation is the slowing of solute velocity relative to the ground water seepage velocity as a result of mass transfer from ground water to the porous medium by adsorption or other chemical processes. The retardation factor is defined as

$$R = v/v_c , \tag{4-12}$$

where v_c is the velocity of the center of mass of the contaminant. Thus, a compound with a retardation factor of 2.0 migrating in ground water traveling at 22 m/y would have an effective VOC migration rate of 11 m/y.

Retardation factors for PCE, TCE, and chloroform were calculated from laboratory-measured soil-water partition coefficients (K_d) according to the following equation:

$$R = 1 + (\rho_b / n) K_d , \tag{4-13}$$

where ρ_b is the bulk density of the aquifer material. These K_d values probably overestimate *in situ* retardation because, under laboratory conditions, the solute has unrestricted access to all sorption sites on the soil particles. In addition, in most measurements the soil sample is sieved to prevent the inclusion of gravel. As gravel content increases, such as along zones of more rapid transport, retardation is lower. With these considerations in mind, we view R values determined from laboratory K_d measurements as upper-bound estimates of retardation. Such values should be considered less conservative, because they represent slower contaminant migration.

For each VOC contaminant, we calculated a best-estimate value of R by taking the average of three values: (1) a minimum value of 1.0, representing the conservative case of no retardation; (2) a maximum value based on laboratory K_d measurements reported by Bishop *et al.* (1989) and discussed in Appendix Q of the RI report (Thorpe *et al.*, 1990); and (3) an expected value of 1.5, which represents an average for all VOCs encountered in the literature review summarized in Appendix Q of the RI report (Thorpe *et al.*, 1990). These best-estimate values are listed in Table 4-2, and range from 1.5 to 3.8. We selected a value of $R = 1.0$, which results in the greatest mobility, for all compounds for the health-conservative simulations.

4.3.5.2.6. Degradation. Degradation is the chemical transformation of a compound, often by stages, into other compounds. For practical purposes, this term is also used to account for loss of VOCs by volatilization and escape to the atmosphere. The rate of transformation of the different VOCs is probably the most difficult parameter to estimate. The literature review by Mallon (1989) indicates that biodegradation of the VOCs does indeed occur, but primarily under anaerobic conditions. Anaerobic degradation products (e.g., 1,2-DCE) have not been observed in the quantities that we would expect if rapid anaerobic degradation were occurring. In addition, we have never detected vinyl chloride, the end product of several possible degradation pathways.

Under aerobic conditions, degradation is expected to occur very slowly, although some research, discussed in Appendix Q of the RI report (Thorpe *et al.*, 1990) suggests half-lives of less than 1 y may be possible. Evidence for abiotic transformation of VOCs in the ground water is even more limited at the present time. Our current understanding of subsurface conditions at the Livermore site suggests that aerobic conditions prevail. For our best estimate of the degradation half-life value, we have conservatively selected a value of 50 y. This transformation rate is sufficient to account for the observed presence of DCEs in ground water, which are either biotransformation products or impurities in the original solvents. No degradation was assumed for the health-conservative simulations.

4.3.5.3. Results

The results for the best-estimate and health-conservative simulations for points A, B, and C in near-, mid-, and far-fields are presented in Tables 4-3 and 4-4, respectively. The three observation fields (i.e., near-, mid-, and far-fields) are listed in order of increasing westerly distance from the Livermore site boundary. We refer to the observation points as potential monitor wells because no real water-supply well would be constructed to capture only the highest concentrations of contaminants. The first column contains the observation point labels. The first letter denotes the observation points along a common flow path; that is, A is due west of the maximum chloroform concentrations, B is west of the maximum TCE and "other VOC" concentrations, and C is west of the maximum PCE concentrations. The second letter of the label indicates its relative distance; that is, N is near-field, M is mid-field, and F is far-field. For example, A-N is the observation point located in the near-field along the simulated flow path of maximum chloroform concentrations.

The compounds simulated are listed in column two, and the simulation results are presented in columns three, four, and five of Tables 4-3 and 4-4. Column three contains 70-y-average concentration values for each compound at each observation point. The maximum 70-y average is listed for the compound of interest at a particular observation point (i.e., chloroform at A points A-N, A-M, and A-F). The values for the other three compounds at a particular point were calculated based on the 70-y period that yielded the maximum average concentration for the compound of interest at that point. If the calculated 70-y average was lower than 0.1 ppb, ND (not detected) was entered into the table (actual laboratory detection limit is 0.5 ppb).

Column four lists the maximum concentrations predicted to occur at each observation point for the compound of interest. The fifth column contains the arrival times, in years, for the predicted maximum concentrations. The arrival times vary for different compounds and observation points within an observation field, because the distance between a compound's area of current maximum concentration and the observation field varies for each compound.

As indicated in Table 4-3, predicted concentrations decrease and arrival times increase with increasing westerly distance from the Livermore site boundary. Concentrations decrease with distance due to degradation and dispersion of the simulated compound (only dispersion for health-conservative simulations because the compounds were simulated as nondegrading).

The health-conservative simulations (Table 4-4) yielded consistently higher predicted maximum concentrations with shorter arrival times than the best-estimate simulations. The faster advection rate in conjunction with no retardation (R = 1.0) produced shorter arrival times in the health-conservative simulations. The higher predicted concentrations are also a result of the higher estimated available mass, lower dispersivity values, and treating the compounds as nonreactive and nonsorptive. The longer transport times of the best-estimate simulations allowed for considerable degradation to occur, resulting in low predicted concentrations. The arrival times of the best-estimate simulations are, in fact, so far into the future that the results must be viewed judiciously. The assumption that the hydrogeologic conditions, particularly the hydraulic gradient, will remain constant for a few hundred years from the present may be transgressed.

The health-conservative simulations also yield higher maximum 70-y averages than the best-estimate simulations for nearly all compounds simulated. Only at the near-field PCE observation

Table 4-3. PLUME results; best-estimate simulations. Predicted VOC concentrations in potential monitor wells downgradient of the Livermore site.

		Best estimate result		
Observation point	Compound[a]	70-y average[b] concentration (ppb)	Maximum concentration (ppb)	Arrival time of maximum concentration (y)
Near-field[c]				
A-N	*Chloroform*	47	90	60
	PCE	ND		
	TCE	27		
	"Other VOCs"	7.4		
B-N[d]	Chloroform	3.2		
	PCE	2.0		
	TCE	150	200	140
	"Other VOCs"	12		
B-N[e]	Chloroform	6.7		
	PCE	1.3		
	TCE	130		
	"Other VOCs"	15	20	120
C-N	Chloroform	5.8		
	PCE	660	790	25
	TCE	25		
	"Other VOCs"	29		
Mid-field[c]				
A-M	*Chloroform*	8.3	12	165
	PCE	ND		
	TCE	0.5		
	"Other VOCs"	0.5		
B-M[d]	Chloroform	ND		
	PCE	0.5		
	TCE	12	14	290
	"Other VOCs"	1.0		
B-M[e]	Chloroform	0.3		
	PCE	0.9		
	TCE	9.0		
	"Other VOCs"	1.4	1.7	255
C-M	Chloroform	ND		
	PCE	8.2	9	280
	TCE	2.2		
	"Other VOCs"	0.4		

Table 4-3. (Continued)

Observation point	Compound[a]	Best estimate result: 70-y average[b] concentration (ppb)	Best estimate result: Maximum concentration (ppb)	Best estimate result: Arrival time of maximum concentration (y)
Far-field[f]				
A-F	*Chloroform*	1.5	2	270
	PCE	ND		
	TCE	ND		
	"Other VOCs"	ND		
B-F[d]	Chloroform	ND		
	PCE	ND		
	TCE	1.0	1	440
	"Other VOCs"	0.1		
B-F[e]	Chloroform	0.1		
	PCE	ND		
	TCE	0.8		
	"Other VOCs"	0.2	0.2	390
C-F	Chloroform	ND		
	PCE	0.1	0.2	525
	TCE	ND		
	"Other VOCs"	ND		

ND—Not detected (concentration below 0.1 ppb).

N/A—Not applicable.

[a]The italicized compound is the compound of interest at that point.

[b]Values calculated from 70-y period that yielded maximum 70-y average for compound of interest.

[c]Under the best-estimate scenario, receptor wells completed in permeable sediments directly in the paths of VOC plumes in the near- and mid-field zones are considered improbable.

[d]Concentrations experienced during the time period of the greatest 70-y average TCE concentration.

[e]Concentrations experienced during the time period of the greatest 70-y average "other VOCs" concentration as the portion of the plume containing carbon tetrachloride passes the observation point.

[f]The far-field wells are co-located with municipal wells in central Livermore. The predicted 70-y average concentrations presented here are reduced by a factor of ten to account for dilution processes (see text).

point (C-N) were the 70-y health-conservative averages lower for PCE and "other VOCs" than for the best-estimate simulation values. The lower PCE 70-y average concentration is a result of the proximity of the PCE "hot spot" to its observation point, which results in a very short arrival time of the maximum predicted concentration (Table 4-3). Although the health-conservative prediction for maximum PCE concentration is greater than the best-estimate predicted maximum concentration, the transport time is so short that the "hot spot" does not disperse significantly and passes the observation point very quickly, resulting in a lower 70-y average. The same is true for the lower "other VOCs" 70-y average concentration. Higher initial concentrations near (and to

Table 4-4. PLUME results; health-conservative simulations. Predicted VOC concentrations in potential monitor wells west of the Livermore site.

		Health-conservative result		
Observation point	Compound[a]	70-y average[b] concentration (ppb)	Maximum concentration (ppb)	Arrival time of maximum concentration (y)
Near-field				
A-N	*Chloroform*	55	220	20
	PCE	6.2		
	TCE	260		
	"Other VOCs"	18		
B-N[c]	Chloroform	20		
	PCE	52		
	TCE	470	2,000	35
	"Other VOCs"	42	150	35
C-N	Chloroform	8.2		
	PCE	270	1,000	5
	TCE	62		
	"Other VOCs"	25		
Mid-field				
A-M	*Chloroform*	49	160	60
	PCE	5.9		
	TCE	240		
	"Other VOCs"	18		
B-M[c]	Chloroform	17		
	PCE	52		
	TCE	380	1,200	75
	"Other VOCs"	40	98	70
C-M	Chloroform	7.3		
	PCE	210	670	45
	TCE	58		
	"Other VOCs"	26		

Table 4-4. (Continued)

Observation point	Compound[a]	Health-conservative result: 70-y average[b] concentration (ppb)	Health-conservative result: Maximum concentration (ppb)	Health-conservative result: Arrival time of maximum concentration (y)
Far-field				
A-F	*Chloroform*	45	120	100
	PCE	6.1		
	TCE	230		
	"Other VOCs"	18		
B-F[c]	Chloroform	16		
	PCE	49		
	TCE	340	900	110
	"Other VOCs"	35	76	110
C-F	Chloroform	6.9		
	PCE	170	460	80
	TCE	56		
	"Other VOCs"	24		

[a]The italicized compound is the compound of interest at that point.

[b]Values calculated from 70-y period that yielded maximum 70-y average for compound of interest.

[c]Under the health-conservative case, the maximum 70-y average concentrations for TCE and the "other VOCs" occurred at the same time for this observation point.

the east of) point C-N are reflected in the short arrival time of the maximum concentration of the "other VOCs" health-conservative results. Again, the short transport time allows less dispersion of the peak concentrations and results in a lower 70-y average for the "other VOCs" in the health-conservative simulation.

Tables 4-5 and 4-6 present results for the D well locations south of East Avenue. These locations coincide with existing private wells that are near, but not directly in, the flow path of the existing VOC plumes. As these tables indicate, only point D-2, which is closest to East Avenue, is predicted to have any detectable concentrations of VOCs. This point coincides with existing well 14C2, operated by Wente Bros. Winery. Well 14C2 is an agricultural well that is gravel-packed from 9 to 58 m. Unlike a monitor well, this well is likely to have lower concentrations of VOCs than predicted by the model, due to dilution with ground water from uncontaminated zones. Furthermore, the assumption of due westward flow from the Livermore site probably overestimates the actual concentrations, because the flow direction is likely to be more northerly in this area, away from East Avenue.

Similarly, if VOCs reach actual municipal supply wells in the central Livermore area, the concentration of VOCs in the water extracted from these wells will probably be much lower than the levels in monitor wells for several reasons. Domestic and municipal supply wells are

Table 4-5. Predicted VOC concentrations in potential monitor wells co-located with existing domestic and agricultural wells south of East Avenue, best estimate simulations.

		Best estimate simulation		
Observation point	Compound[a]	70-y average[b] concentration (ppb)	Maximum concentration (ppb)	Arrival time of maximum concentration (y)
D-1	Chloroform	ND	0.2	60
	PCE	ND	ND	N/A
	TCE	ND	ND	N/A
	"Other VOCs"	ND	ND	N/A
D-2	Chloroform	1	2	100
	PCE	5	6	150
	TCE	2	2	200
	"Other VOCs"	1	1	100
D-3	Chloroform	ND	ND	N/A
	PCE	ND	ND	N/A
	TCE	ND	ND	N/A
	"Other VOCs"	ND	ND	N/A

ND—Not detected (concentration below 0.1 ppb).

N/A—Not applicable.

typically screened over much longer intervals than monitor wells, which means that any contaminated water entering the well from one zone will be diluted by clean water derived from other vertical zones. In particular, the CWSC municipal wells near central Livermore (9L1, 9P1, 9Q1, and 16B1) are completed over 100 m, whereas, near the distal margins of the VOC plume, the thickness of permeable sediments containing VOCs is typically less than 10 m. Secondly, the CWSC wells are located in the Mocho II subbasin and probably receive the majority of their water from the Arroyo Mocho drainage area rather than the Mocho I subbasin. Thirdly, the CWSC wells produce a cone of drawdown sufficient to induce ground water flow not only from the east, but from the south and west. These factors will combine to reduce the concentrations of VOCs in extracted water to less than one-tenth of the predicted values under the best-estimate case. Quantitative evaluation of these factors is being carried out with a two-dimensional numerical model of the regional ground water system. Preliminary results, which will be reported soon, indicate that the 10-fold reduction is reasonable.

4.3.6. Gas Spill Migration Analysis

In this section, we evaluate the potential for gasoline constituents (particularly benzene), to migrate from the Gasoline Spill Area, in the south-central area of LLNL to offsite areas.

Table 4-6. Predicted VOC concentrations in potential monitor wells co-located with existing domestic and agricultural wells south of East Avenue, health-conservative simulations.

		Health-conservative simulation		
Observation point	Compound[a]	70-y average[b] concentration (ppb)	Maximum concentration (ppb)	Arrival time of maximum concentration (y)
D-1	Chloroform	ND	0.2	20
	PCE	ND	ND	N/A
	TCE	ND	0.3	35
	"Other VOCs"	ND	ND	N/A
D-2	Chloroform	3	6	40
	PCE	12	40	25
	TCE	12	40	55
	"Other VOCs"	4	10	50
D-3	Chloroform	ND	0.1	60
	PCE	ND	ND	N/A
	TCE	ND	0.2	70
	"Other VOCs"	ND	ND	N/A

ND—Not detected (concentration below 0.1 ppb).

N/A—Not applicable.

4.3.6.1. Background

The gasoline spill consisted of approximately 65,000 L of leaded gasoline, lost between about 1952 and 1979 from the southernmost of four underground fuel tanks located near former Building 403. Calculations of hydrocarbon mass based on chemical analyses of saturated and unsaturated sediment and ground water indicate that as of 1988, about 23,000 L existed in the vadose zone, about 38,000 L was present in saturated sediments, and about 380 L was dissolved in ground water. All four tanks were taken out of service in 1979. Subsequent investigations indicated that the leak had occurred at either the western edge of the southernmost tank and/or in a distribution line for the tank (Nichols *et al.*, 1988).

4.3.6.2. Summary of the Local Hydrogeology

The hydrogeology of LLNL and vicinity has been discussed in detail in Section 3 of the RI report (Thorpe *et al.*, 1990); further background information is available in the References section (Stone *et al.*, 1982; Stone and Ruggieri, 1983; Carpenter *et al.*, 1984; Weiss Associates, 1985; and Dresen and Hoffman, 1986). Additional local hydrogeologic data for the Gasoline Spill Area have been published in Carpenter (1984), O. H. Materials (1985), Dresen *et al.* (1986), and various monthly progress reports of the LLNL Ground Water Project.

Depth to ground water in the Gasoline Spill Area ranges from about 27 to 30 m. Figure 4-25 is a ground water level map constructed from water table elevations from 23 wells that were measured by LLNL during June 13–16, 1989. Water levels were contoured from wells completed in the second and third water-bearing zones [i.e., between about 35 and 45 m in depth]. The majority of wells in the area are completed in the second and third zones, which are more transmissive than the uppermost zone. Water levels in the second and third water-bearing zones are very similar (Nichols *et al.*, 1988).

The horizontal hydraulic gradient in the Gasoline Spill Area is generally quite low, ranging from about 0.001 to 0.006 m/m, with a broad north-south ground water divide extending from the area around GSW-215 southward through the leak point to the area near GSW-1A. Ground water on the east flank apparently flows toward the east-southeast, and ground water on the west flank flows to the southwest.

4.3.6.3. Current Distribution of Contaminants

The distribution of fuel hydrocarbons in the vadose and saturated zones of the Gasoline Spill Area is discussed in detail in Dresen *et al.* (1986) and Nichols *et al.* (1988). Fuel hydrocarbons apparently have not migrated in ground water in a preferred direction from the leak point, and most of the mass of the fuel hydrocarbons in ground water is located within about 80 m of the leak point near GSW-16 (Nichols *et al.*, 1988).

Benzene was chosen as an indicator compound for estimation of whether gasoline constituents may migrate beyond DOE property. Benzene is more soluble than most gasoline constituents (solubility = 1,780 mg/L) (Mallon, 1989) and is a known carcinogen. The areal distribution of benzene in ground water, shown in Figure 4-26, is very similar to the distribution of total fuel hydrocarbons (TFHCs) and total aromatic hydrocarbons, which include benzene, ethyl benzene, toluene, and xylenes (BETX) (Nichols *et al.*, 1988). In addition, laboratory and field data exist for the parameters required to estimate the transport and fate of benzene in ground water (i.e., retardation factor and degradation half-life).

4.3.6.4. Benzene Migration Modeling

The potential for transport of benzene beyond DOE property was analyzed using the PLUME code and the following input parameters:

4.3.6.4.1. Mass Estimate. Mayrsohn *et al.* (1978) calculated that regular leaded gasoline produced in 1978 contained an average 1.1% benzene by weight. To be conservative, we assume that the estimated 65,000 L of spilled gasoline contained 2% benzene by weight. The density of gasoline is approximately 0.74 kg/L (Bolz and Tuve, 1973), so 65,000 L contain about 48,000 kg gasoline, or conservatively, approximately 1,000 kg benzene.

4.3.6.4.2. Ground Water Velocity. Although the local ground water gradient is quite low and the local flow directions are divergent, the regional potentiometric surface indicates a general southwest to westerly flow direction from the Gasoline Spill Area. As a conservative approach, we assume that the transport of hydrocarbons in ground water will be toward the

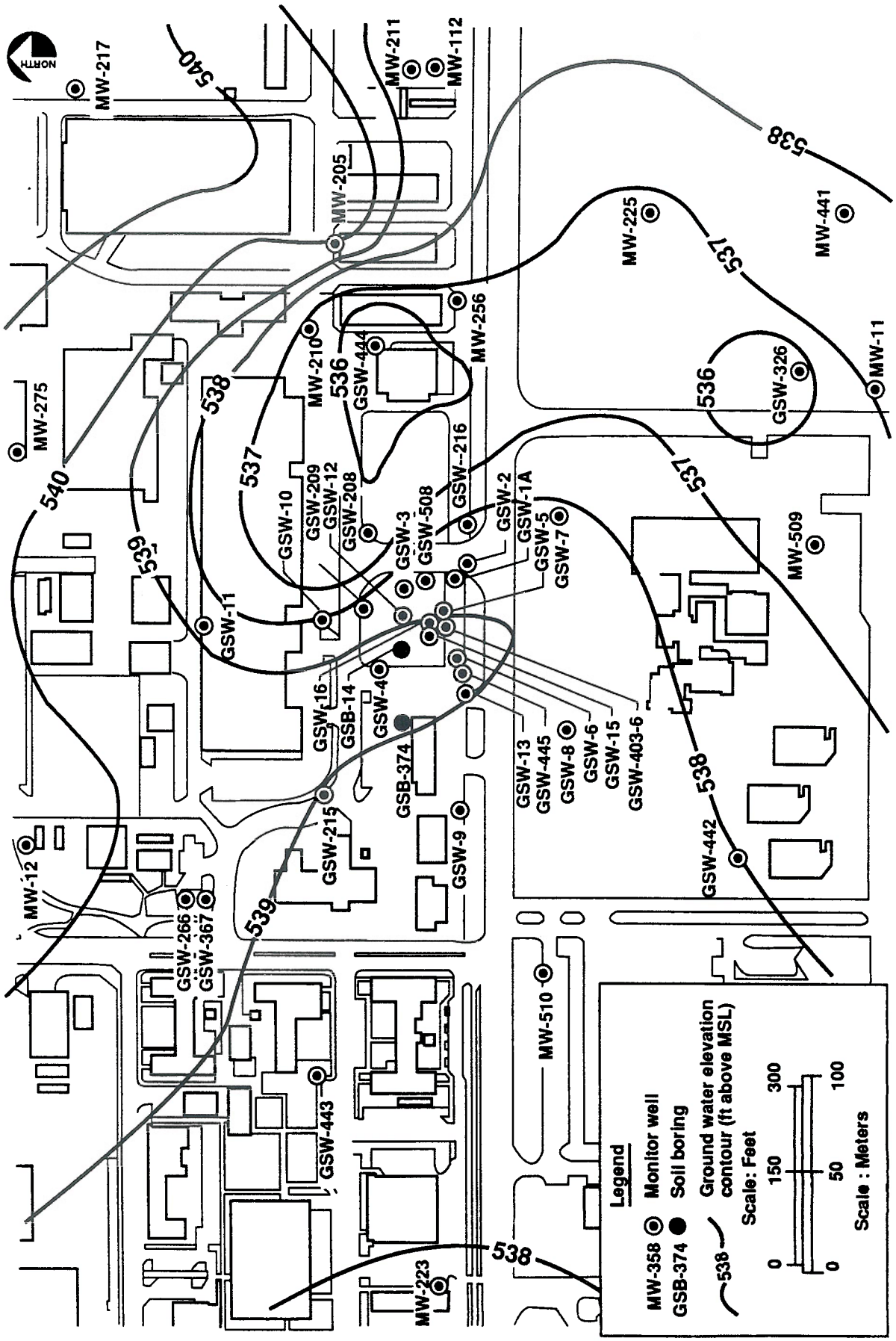

Figure 4-25. Ground water elevation contour map, June 1989, Gasoline Spill Area.

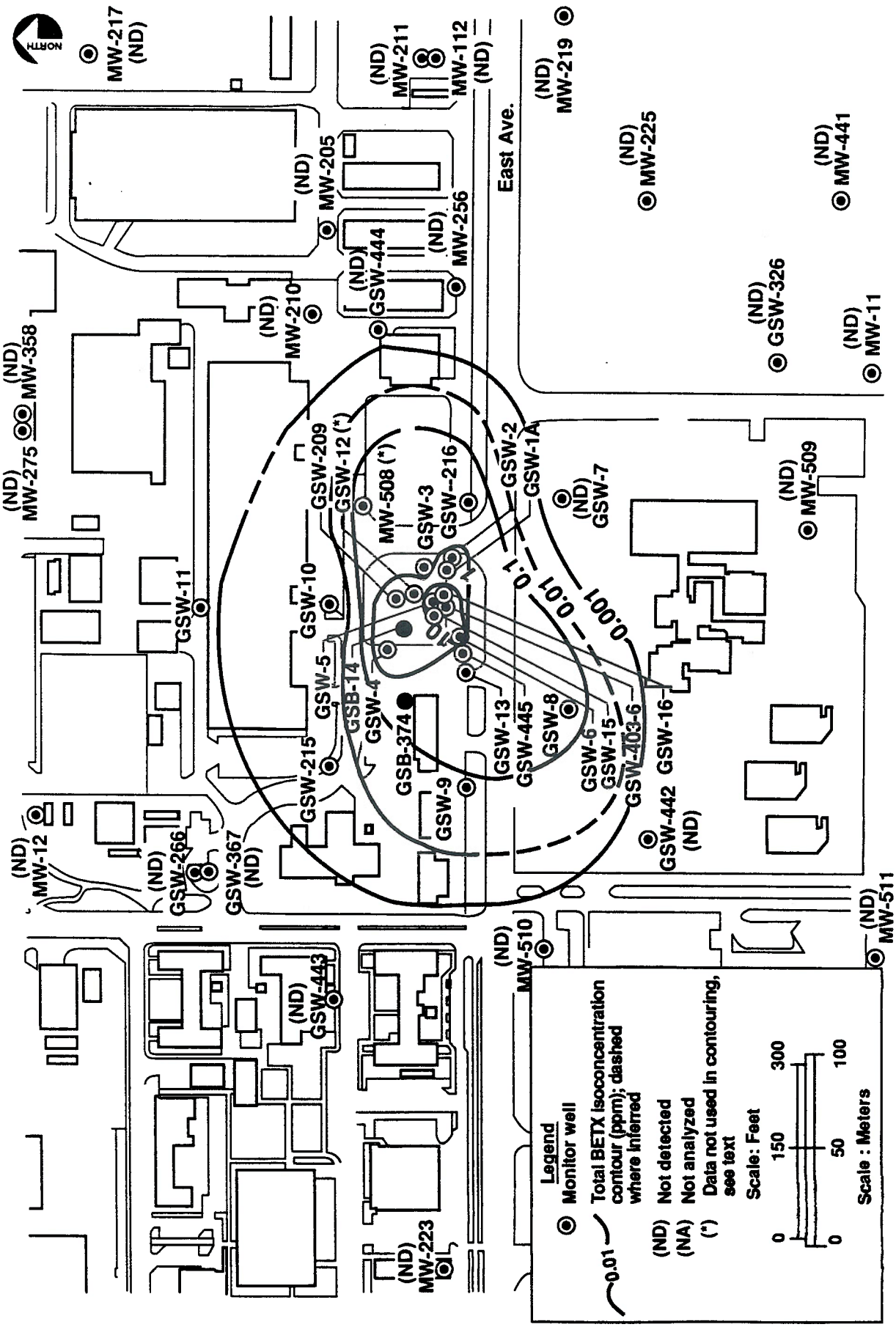

Figure 4-26. Isoconcentration contour map of benzene in ground water, March 1989.

nearest conceivable downgradient, non-DOE property, about 1,300 m west of the Gasoline Spill Area.

The ground water velocity in the Gasoline Spill Area is controlled by the composition and structure of the saturated sediments and the local hydraulic gradient. The present-day distribution of hydrocarbons indicates that the plume has not migrated significantly; that is, the isoconcentration contours remain relatively close to and nearly concentric about the gasoline leak point. However, as a conservative estimate of the ground water velocity between the Gasoline Spill Area and the DOE property boundary at Vasco Road, we calculated a distance-weighted effective ground water velocity of 19 m/y. This value is based on the best-estimate site-wide velocity of 22 m/y, determined previously, and the estimated ground water velocity within the Gasoline Spill Area of 8 m/y. The velocity of 8 m/y in the Gas Spill Area is based on a local gradient of approximately 0.001 and an effective hydraulic conductivity of 6.4 m/d.

4.3.6.4.3. Retardation. Seip *et al.* (1986) measured the retardation of benzene in laboratory column experiments using three different Norwegian soils. Using a soil sample composed of 97.3% sand and containing 0.2% organic material, Seip *et al.* obtained a retardation factor for benzene of 1.34 (see Mallon, 1989). Based on this information, we assume a retardation factor of one (i.e., no retardation) for the transport estimate.

4.3.6.4.4. Degradation. The degradation process for benzene is described in Mallon (1989). It ultimately degrades to water, carbon dioxide, and methane, with various other intermediate compounds. *In situ* or estimated degradation half-life values range from 0.2 to less than 1.2 y. Wilson *et al.* (1986) measured a transformation half-life of 0.63 y in laboratory experiments. However, biodegradation of benzene usually occurs at lower concentrations. In the immediate vicinity of the gasoline leak point, the concentrations of benzene may be too high for biodegradation to occur. Within about 60 m of the leak point, however, the concentrations are probably low enough that microbial degradation may become significant. Based on these values, a conservative degradation half-life of 5 y is assumed for the purpose of our screening analysis.

4.3.6.4.5. Other Parameters. The remaining parameters for this analysis, including porosity, aquifer thickness, and dispersivity, were the same as those used for the previous VOC migration calculations. A porosity of 0.30 and an aquifer thickness of 5 m were assumed, and longitudinal and transverse dispersivities were assigned values of 10 m and 1 m, respectively. Only the health-conservative values of dispersivity were used because of the smaller dimensions of the migration pathway.

4.3.6.5. Results

Using the conservative assumptions described above (d = 1330 m, R = 1, and v = 19 m/y), we obtained a transport time of about 68 y for benzene to reach the DOE property boundary at Vasco Road. Based on a degradation half-life of 5 y, the concentrations in ground water at the DOE property boundary at that time will be about 1 ppb. We conclude, therefore, that benzene and associated gasoline constituents are not likely to migrate to any receptor off DOE property at concentrations above the State MCL of 1 ppb (see Table 3-5 in Section 3 of this document). In addition, these estimates are based on the assumption that no remediation of the gasoline spill will be conducted, when in fact, the current pilot remediation effort has already reduced the total contaminant mass.

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5. Baseline Risk Assessment

D. W. Layton, T. E. McKone, L. C. Hall, and K. T. Bogen

5.1. Public Health Evaluation

The Baseline Public Health Assessment (BPHA) addresses the future public-health risks that could exist if no cleanup were attempted at the LLNL site. Under this scenario, we assume that no attempt is made to either mitigate or prevent exposures to toxic substances. In essence, the assessment serves as a baseline case that can be used to compare the relative effectiveness of alternative remediation strategies in reducing public-health risks. It is important to keep in mind, however, that the DOE, LLNL, and environmental regulatory agencies are dedicated to the remediation of contaminated soils, sediments, and ground water at the LLNL site. Given this perspective, the BPHA provides the information that is required to evaluate the benefits of cleanup alternatives.

Pertinent information on the content and preparation of health assessments at Superfund sites is contained in *Guidance for Conducting Remedial Investigations and Feasibility Studies Under CERCLA* (U.S. EPA, 1988a), the *Superfund Exposure Assessment Manual* (U.S. EPA, 1988b), and the *Superfund Public Health Evaluation Manual** (U.S. EPA, 1986a). The primary components of the BPHA are outlined in Figure 5-1.

The first task of the baseline risk assessment was to evaluate the hazards posed by the contaminants detected in the environmental media (e.g., soils, sediments, and ground water) at the site. This evaluation, described in Section 3 of this document, identified those contaminants that *potentially* pose the greatest risks to human health. Once the relevant set of contaminants was defined, we simulated their migration in ground water to selected receptor wells (see Section 4 of this document). This section uses those results to assess the exposure and resultant public health risk.

In the exposure assessment, which is described in Section 5.1.1, we examine the pathways by which an individual can come into contact with contaminants at or near the LLNL site via inhalation, ingestion, and dermal absorption. In the dose assessment, we estimate of the mass of a nonradioactive substance (or the energy of a radioactive substance) deposited in a human body or selected organs and tissues as a result of exposures. Dose-response functions relate the predicted doses to health effects, described in Section 5.1.2. Each component of the risk-assessment process involves uncertainties. Some uncertainties are difficult to quantify because of data gaps or our ignorance regarding an underlying process (e.g., carcinogenesis). Other uncertainties relate to the variability among individuals, arising from differences in physiology and lifestyle. Consequently, we have made assumptions and developed scenarios to reflect such uncertainties.

* The most recent guidance document from the EPA for preparing a baseline public health assessment (U.S. EPA, 1989a), which replaces the *Superfund Public Health Evaluation Manual* (U.S. EPA, 1986a), was not published until well after the draft version of this BPHA was submitted for review.

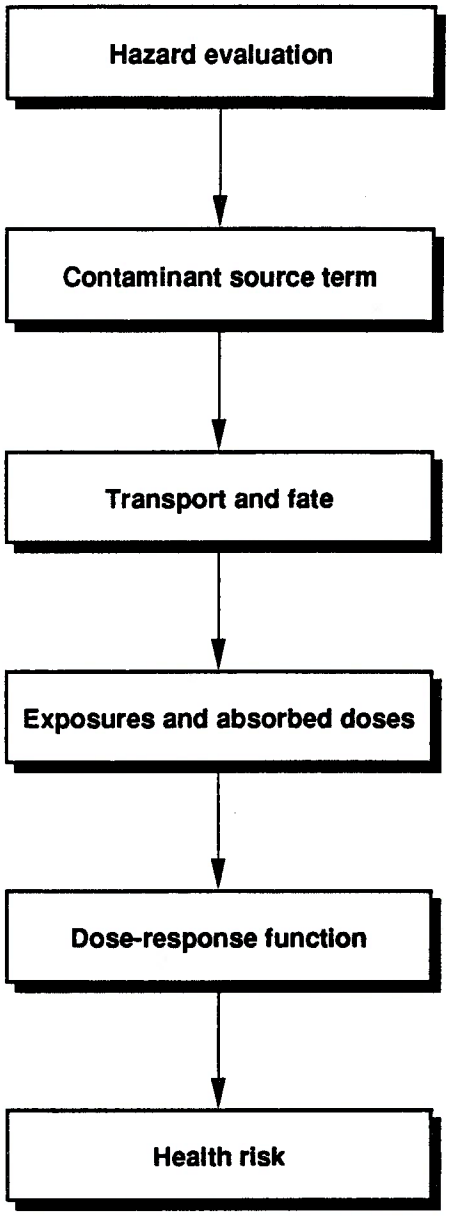

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Figure 5-1. Major components of the risk assessment process.

5.1.1. Exposure and Dose Assessments

Our screening analyses in Section 3 of this document indicated that VOCs in ground water constitute the greatest potential risk from the standpoint of public health. Accordingly, we examined different aspects of the ground water exposure pathway. These aspects included current uses of ground water to the west of the LLNL site, locations of existing wells, and the potential for new wells in that area. We then predicted concentrations of selected VOCs in potential monitor wells that draw water from permeable sediments in the path of the ground water plume(s) containing the VOCs of concern. Here, we estimate exposures to contaminated well waters via the primary ground water uses, which are domestic and irrigation uses.

Figure 5-2 shows the principal pathways we considered in our exposure assessment. For domestic uses, we considered three forms of water-based exposure:

- Direct ingestion of water for drinking.
- Inhalation of VOCs volatilized from showers.
- Dermal uptake of VOCs in bath water.

For irrigation uses, we considered two pathways:

- Inhalation of VOCs that volatilize from sprinklers.
- Ingestion of vegetables from home gardens irrigated with water containing VOCs.

To calculate potential exposures to VOCs via the various pathways, we must translate predicted VOC concentrations in water to daily intakes, based on human factors affecting exposure (e.g., daily amount of water consumed and breathing rate) as well as environmental transport factors (e.g., uptake in crops and transfer efficiency of VOC from water to air). We present our methods, assumptions, and supporting data used to estimate water-based exposures to VOCs in ground water in the following paragraphs.

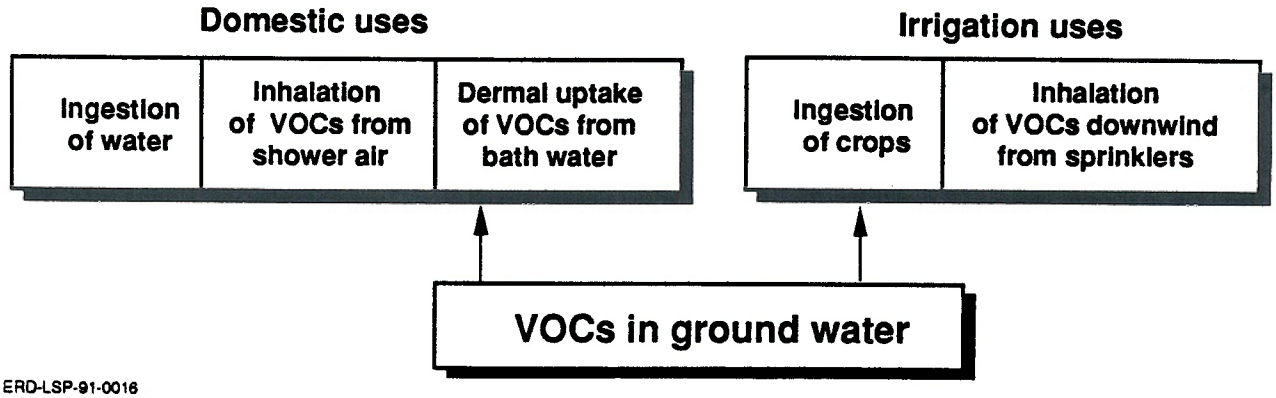

Figure 5-2. Overview of the water-based exposure pathways considered in the public health assessment.

5.1.1.1. Water-Based Exposure Pathways Associated with Domestic Water Uses

Our basic approach to estimate exposure for each water-based pathway is to calculate a unit pathway-exposure factor (PEF). A PEF converts a predicted or measured concentration of a VOC in water into an equivalent, average lifetime exposure. (We used a similar approach in Section 3 of this document to calculate soil-based exposures to various substances.) The lifetime exposure e_i (in units of mg/kg-d) via a specific pathway i is

$$e_i = F_i C_W , \tag{5-1}$$

where

F_i = pathway exposure factor (L/kg-d),

C_W = concentration of VOC in water (mg/L), and

i = applicable pathway (where 1 = ingestion, 2 = inhalation, and 3 = skin absorption).

Because we are interested in the equivalent lifetime exposure for an individual, we calculated exposure factors for three consecutive age categories (infant, child, and adult). The overall exposure factor F_i is estimated as the weighted sum of the pathway-exposure factors, f_i(age group), for each of the three age categories, as follows:

$$F_i = \frac{2}{70} f_i(\text{infant}) + \frac{14}{70} f_i(\text{child}) + \frac{54}{70} f_i(\text{adult}) . \tag{5-2}$$

In this expression, the factors 2/70, 14/70, and 54/70 reflect the fractions of a 70-y lifespan associated with each age category. We also assumed that an individual uses contaminated water for 70 y at a single location (e.g., a residence supplied with well water). In addition, we calculated pathway dose factors (PDFs) for each VOC to account for metabolism. A PDF for a pathway is calculated as $F_{di} = aF_i$, where a is the fraction of VOC exposure that is metabolized. This must be done because the carcinogenicity of PCE, TCE, and chloroform is thought to be related to the formation of reactive metabolites. Consequently, an estimate of an effective or metabolized dose is needed to calculate cancer risk. Such doses are discussed in Section 5.1.2.

5.1.1.1.1. Water Ingestion. For the water-ingestion pathway (i = 1), we obtained the value of f_i for each age category by dividing daily water intake by body weight. The ratio of water intake to body weight for each age group comes from Table 5-1, which includes only tap water and water-based drinks, and Table 5-2 (body weights). We also made the conservative assumption that all fluids consumed by members of a household with contaminated water are at the same concentration level. Thus, F_1 is given by

$$F_1 = \frac{2}{70} \times 0.038 + \frac{14}{70} \times 0.020 + \frac{54}{70} \times 0.017$$

$$= 0.018 \text{ mg/kg-d per mg/L (or L/kg-d)} . \tag{5-3}$$

The estimated value of F_1 is less than the value obtained under the historic assumption that intake of total fluids over a lifetime approximates 2 L/70 kg-d or 0.028 (mg/kg-d)/(mg/L). This

Table 5-1. Water intakes for infants, children, and adults (Yang and Nelson, 1986).

	Infants; young children (< 4y) (L/d)	Older children; teens (4 to 19 y) (L/d)	Adult (L/d)
Tap water	0.39	0.60	0.68
Water-based drinks[a]	0.05	0.15	0.53
Total water intake	0.44	0.75	1.21
		(L/kg-d)	
Fluid intake per unit body weight	0.038	0.020	0.017

[a]Includes tea, coffee, and juices made from tap water.

Table 5-2. Human body weight and surface area by age for males and females (U.S. EPA, 1989b).

Age (y)	Mass (kg)	Surface area (m^2)
Infants; young children (< 3 y)	11.6	0.59
Older children; teens (3 to 18 y)	38.0	1.2
Adult (>18 y)	71.8	1.8

difference results because we have omitted intakes from milk, soft drinks, and other non-tap-water beverages. One final assumption is that *ingested* VOCs (in contrast to VOCs inhaled or absorbed through the skin) are completely metabolized. This assumption is made because of the "first-pass" metabolism that occurs after these VOCs are absorbed into blood draining the gastrointestinal tract and transported through the hepatic portal vein directly to the liver, where they are metabolized (Bogen *et al.*, 1988).

*5.1.1.1.2. **Inhalation Exposure.*** Several researchers (Prichard and Gesell, 1981; Cothern *et al.*, 1984; Andelman, 1985; and Foster and Chrostowski, 1986) have addressed the relative contribution of the respiratory pathway to overall human exposures from VOCs in tap water. All have found that volatile compounds in water supplies can result in inhalation exposures that are comparable to the ingestion pathway.

McKone (1987) developed a model that describes the daily concentration profile of VOCs within the various components of the indoor air volume of a dwelling. The model divides the indoor air volume into three compartments: the shower/bath stall, the bathroom, and the household volume. We used this model to calculate a PEF that corresponds to the use of contaminated water in the indoor environment. The estimated PEF corresponds to the average lifetime daily exposure to an individual living in a typical California household. Breathing rates for infants, children, and adults are 19, 19, and 13.5 L/kg-h (McKone, 1987; Bogen *et al.*, 1988).

For the typical home, we assumed that the household has four occupants and uses 900 L/d of water contaminated with PCE, TCE, chloroform, or 1,1-DCE. We also assumed that each of those VOCs has water-to-air transfer properties similar to those of radon-222. The ratios of the mass-transfer coefficients of the VOCs to the mass-transfer coefficient of radon are approximately 0.7 (McKone, 1987).

We estimated the time-dependent concentration profile of a VOC in the shower stall, bathroom, and household air, and the resulting effective lifetime doses, using the following assumptions:

- Occupants spend 100% of their time in the house from 11:00 p.m. to 7:00 a.m.
- The bathroom is used for showers/baths from 7:00 a.m. to 8:00 a.m.
- Each adult and child spends 20 min in the bathroom during the period from 7:00 a.m. to 9:00 a.m.
- Each adult spends 10 min in the shower or bath.
- Adults spend 25% of the time from 7:00 a.m. to 11:00 p.m. in the house.
- Children spend an average of 20 min/wk in showers or baths.
- Children spend 60% of the time between 7:00 a.m. and 11:00 p.m. in the house.
- Infants spend 100% of their time in the house and 2% of that time in a bathroom.
- 100% of the VOCs inhaled in alveolar air is available for pulmonary uptake. (Alveolar ventilation rates are approximately 30% of the total breathing rates.)

Using this model, we estimate that the pathway-exposure factor for inhalation, F_2, in typical households is

$$F_2 = 0.024 \text{ mg/kg-d per mg/L (L/kg-d)} . \quad (5\text{-}4)$$

This estimate is based on the assumption that an adult showers every day and that children bathe every second day. Table 5-3 summarizes the relative contribution to the PEF, F_2, from each age category and household compartment. For adults or children who take baths instead of showers, these numbers are likely to be reduced somewhat. We have not examined the extent of reduction that taking baths in place of showers would give. Table 5-3 reveals that for adults, exposures in the shower and bathroom are the major contributors to indoor inhalation exposures attributable to contaminated water. Although the assumption that adults spend 25% of the time from 7:00 a.m. to 11:00 p.m. in the house may seem low, we believe this is a plausible value for a typical adult. Such an adult would spend 10 h/d in work and travel, leaving 6 h of leisure time, of which we assume roughly two-thirds is actually spent in the home. Furthermore, we assume that roughly three-fourths of all adults work outside the home. Finally, we have found that the PEF is insensitive to this occupancy factor (see McKone, 1987).

The PEFs for PCE, TCE, and chloroform must be adjusted to derive estimates of PDFs. (The health risks for other VOCs are based on exposure rate, not effective metabolized dose, and, therefore, no adjustment is necessary.) In this regard, Bogen and McKone (1988) estimated that between 3.7 and 47% of the PCE present in alveolar air is metabolized by humans. We used the

Table 5-3. Percent contribution to the lifetime average exposure factor F_2 (inhalation) from specific age and household exposures.

Age	Household exposure	Contribution (%)
Adult	Shower	51
	Bathroom	20
	Remainder of house	7.9
Child	Shower	5.2
	Bathroom	7.2
	Remainder of house	7.0
Infant	Bathroom	0.17
	Remainder of house	1.2
		100

geometric mean of this range (i.e., 13%) to calculate the PDF for PCE. The value for PCE is considerably lower than the 72% estimated for TCE (Bogen *et al.*, 1988) and 68% for chloroform (Bogen *et al.*, 1989). The F_{d2} parameters are therefore

$$F_{d2}\,(\text{PCE}) = 0.13 \times 0.024 = 0.0031\ \text{L/kg-d}\,, \tag{5-5}$$

$$F_{d2}\,(\text{TCE}) = 0.72 \times 0.024 = 0.017\ \text{L/kg-d}\,, \tag{5-6}$$

and

$$F_{d2}\,(\text{chloroform}) = 0.66 \times 0.024 = 0.016\ \text{L/kg-d}\,. \tag{5-7}$$

5.1.1.1.3. Dermal Absorption. We reviewed the literature on absorption rates of volatile solvents having direct contact with the skin to estimate the likely value of dermal absorption from normal daily use of contaminated water. Over the last 20 y, several investigators have examined the transport of dissolved chemicals through skin (Stewart and Dodd, 1964; Riihimaki and Pfäffli, 1978; Brown *et al.*, 1984; Cothern *et al.*, 1984; Scheuplein and Blank, 1971; Bronaugh, 1985; Foster and Chrostowski, 1986). Although it is a complex process, dermal uptake of compounds in aqueous solution occurs mainly by passive diffusion through the stratum corneum (the outer layer of keratinized cells of the epidermis).

We assumed that dermal exposure occurs during bathing and showering. To determine the PEF for dermal absorption, we made the following simplifying assumptions:

- Resistance to diffusive flux through layers other than the stratum corneum is negligible.
- Steady-state diffusive flux is proportional to the concentration difference between the skin surface and internal body water.

- An adult spends about 10 min in a bath or shower each day.
- During bathing, roughly 80% of the skin is in contact with water; during showers, roughly 40% of the skin is in contact with water.
- Children and infants spend approximately 1 h/wk bathing or swimming (U.S. NRC, 1977).

The exposure, e_3, from dermal absorption is given by

$$e_3 = J_S \tau f_S SA , \quad (5\text{-}8)$$

where

J_S = steady-state flux across the stratum corneum (mg/cm^2-h),

τ = duration in the shower or bath (h),

f_S = fraction of the skin surface in contact with water (unitless), and

SA = surface area of the skin (cm^2).

We assume that chemical transport across the skin follows Fick's law. The flux J_S across skin tissue is

$$J_S = K_p \Delta C_{sk} , \quad (5\text{-}9)$$

where

K_p = permeability constant across the stratum corneum L/cm^2-h), and

ΔC_{sk} = concentration difference of the solute across skin (mg/L).

Brown *et al.* (1984) determined that K_p is on the order of 0.001 L/cm^2-h for VOCs. For dilute solutions, ΔC_{sk} is approximately equal to the chemical concentration at the skin surface. However, the concentration at the skin surface is not necessarily the same as the concentration in the water supply. For showers, we assume that $C_{sk} = C_w$, the VOC concentration in tap water. However, for bathing, in which water stands for a period of time, we use C_{sk} equal to the average water concentration over the period of the bath. Assuming an exponential loss of one-half of the dissolved VOC over a period of 10 min, we obtain $C_{sk} = 0.72\ C_w$ during a 10-min bath and $C_{sk} = 0.54\ C_w$ during a 20-min bath (Foster and Chrostowski, 1986).

Using the above relations, we obtain a lifetime equivalent exposure factor for dermal absorption as follows:

$$F_3 = K_p\left[\frac{2}{70}\left(\tau f_S \frac{SA}{BW} C_{sk}\right)_{infant} + \frac{14}{70}\left(\tau f_S \frac{SA}{BW} C_{sk}\right)_{child} + \frac{54}{70}\left(\tau f_S \frac{SA}{BW} C_{sk}\right)_{adult}\right], \quad (5\text{-}10)$$

in which BW represents body weight in kilograms, SA represents surface area in square centimeters, and τ is the duration of bath or shower. With water containing 1 mg/L and a 10-min bath, the term $\tau f_S C_{sk}$ is on the order of 6 min-mg/L; for a 10-min shower, this term is roughly

4 min-mg/L; for a 15-min shower, the term is 6 min-mg/L. Thus, given the uncertainty in the overall exposure estimate, we consider a 10- or 20-min bath as an appropriate representation for the best estimate of dermal absorption during both baths and showers.

Based on data from Table 5-2, the ratios SA/BW for adults, children, and infants are 250, 320, and 510 cm^2/kg, respectively. Substituting the appropriate values into Eq. (5-10) gives the following estimate of F_3:

$$F_3 = 0.001 \frac{L}{cm^2\text{-h}}\left[\frac{2}{70}\left(0.17 \frac{h}{d} \times 0.80 \times 510 \frac{cm^2}{kg} \times 0.72\, C_w\right)\right.$$

$$+ \frac{14}{70}\left(0.17 \frac{h}{d} \times 0.80 \times 320 \frac{cm^2}{kg} \times 0.72\, C_w\right)$$

$$\left. + \frac{54}{70}\left(0.17 \frac{h}{d} \times 0.80 \times 250 \frac{cm^2}{kg} \times 0.72\, C_w\right)\right]$$

$$= 0.027 \frac{mg}{kg\text{-d}} \text{ per } \frac{mg}{L} \text{ (L/kg-d)} . \qquad (5\text{-}11)$$

Adjusting the values of F_3 to account for metabolism of PCE, TCE, and chloroform, we obtain the following F_{d3} values:

F_{d3} (PCE) = 0.13 × 0.027 = 0.0035 L/kg-d ,

F_{d3} (TCE) = 0.72 × 0.027 = 0.019 L/kg-d ,

and

F_{d3} (chloroform) = 0.66 × 0.027 = 0.018 L/kg-d .

5.1.1.1.4. Summary of Pathway-Exposure Factors. Table 5-4 summarizes the three pathway-dose factors for PCE, TCE, and chloroform, and the pathway exposure factor for the other VOCs. The sums of the individual PDFs for the VOCs range from 0.025 to 0.054 L/kg-d, whereas the PEF for the other VOCs is 0.069 L/kg-d.

The PDF sums are 1.4 to 3 times higher than the PDF for water ingestion alone. For PCE, water ingestion accounts for 73% of the total water-based exposure, followed by dermal absorption (14%) and inhalation (13%). Individual PDF values for TCE and chloroform are quite similar.

Table 5-4. Summary of the pathway dose factors for PCE, TCE, and chloroform and the pathway exposure factor for 1,1-DCE and carbon tetrachloride.

	Pathway			
Contaminant	Water ingestion, F_{d1} (L/kg-d)	Indoor inhalation, F_{d2} (L/kg-d)	Dermal absorption, F_{d3} (L/kg-d)	Totals (L/kg-d)
PCE	0.018	0.0031	0.0035	0.025
TCE	0.018	0.017	0.019	0.054
Chloroform	0.018	0.016	0.018	0.052
Other VOCs[a]	0.018	0.024	0.027	0.069

[a]Used in the exposure assessment for 1,1-DCE and carbon tetrachloride.

5.1.1.2. VOC Exposures Associated with Domestic Uses of Well Water

We used the two contaminant-transport scenarios to predict maximum 70-y average concentrations of VOCs in potential monitor wells downgradient of the LLNL site. Dose rates for PCE, TCE, and chloroform are calculated as

$$D = C_w (F_{d1} + F_{d2} + F_{d3}) , \quad (5\text{-}12)$$

where C_w is the 70-y average concentration of each VOC, and F_{d1}, F_{d2}, and F_{d3} are the unit pathway dose factors discussed earlier. The exposure rate for the other VOCs is calculated in a similar fashion, except that we used the sum of the individual PEFs. Table 5-5 shows the exposure estimates for each contaminant and far-field well. In this table, the best-estimate scenario we have chosen represents the case in which VOCs undergo slow degradation and are retarded due to adsorption onto aquifer materials. Under the best-estimate case, the drilling of closer wells directly in the path of VOC plumes is considered improbable, and hence exposures are not estimated. However, under the health-conservative case, shown in Table 5-6, water-based exposures have been estimated for potential monitor wells in the near- and mid-field areas. Exposures for wells D-1 and D-3 (associated with existing irrigation wells) were not estimated because the estimated maximum 70-y concentrations for each of the VOCs were below the detection limit of 0.1 ppb. Exposures associated with the use of well D-2, representing an irrigation well, are addressed in the following paragraphs.

5.1.1.3. Water-Based Exposure Pathways Associated with Irrigation Uses of Well Water

Two possible exposure pathways relate to the use of well water for irrigation: consumption of fruits and vegetables from a home garden, and inhalation of VOC vapors volatilized from sprinklers irrigating vineyards adjacent to East Avenue. The first exposure pathway could contribute to the overall exposure for members of a household using water containing VOCs. However, for the second pathway associated with irrigation, only those individuals living adjacent to irrigated areas would be exposed to VOCs.

Table 5-5. Water-based exposures to VOCs in municipal wells for the best-estimate scenario.[a]

Far-field well[b,c]	Best-estimate scenario (dose rate in mg/kg-d) PCE	TCE	Chloroform	Other VOCs
A-F	N/A[d]	N/A[d]	7.8×10^{-6}	N/A[d]
B-F[e]	N/A[d]	5.4×10^{-6}	N/A[d]	6.9×10^{-7}
B-F[e]	N/A[d]	4.3×10^{-6}	5.2×10^{-7}	1.4×10^{-6}
C-F	2.5×10^{-7}	N/A[d]	N/A[d]	N/A[d]

[a]In this scenario, VOCs undergo slow degradation and are retarded due to adsorption on aquifer material. Exposures are calculated from the highest 70-y average concentrations presented in Table 4-3.

[b]See Figure 4-5 for well locations. The far-field wells represent municipal wells in central Livermore.

[c]Under the best-estimate scenario, the predicted VOC concentrations for the far-field monitor wells were reduced by a factor of 10 to account for the dilution processes associated with pumping municipal wells (see Section 4.3.5.3). Estimated dose rates presented here were calculated with the adjusted concentrations.

[d]Not applicable. Predicted concentrations were below the detection limit of 0.1 ppb.

[e]This observation point had the highest 70-y concentrations for TCE and the "other VOCs." However, the predicted concentrations for the two VOCs occur at different times; thus, exposure estimates are reported separately.

Table 5-6. Water-based exposures to VOCs in potential monitor wells for a health-conservative scenario.[a]

Far-field well[b]		Health-conservative scenario (dose rate in mg/kg-d) PCE	TCE	Chloroform	Other VOCs
Near-field	A-N	1.5×10^{-4}	1.4×10^{-2}	2.9×10^{-3}	1.2×10^{-3}
	B-N	1.3×10^{-3}	2.5×10^{-2}	1.0×10^{-3}	2.9×10^{-3}
	C-N	6.6×10^{-3}	3.3×10^{-3}	4.3×10^{-4}	1.7×10^{-3}
Mid-field	A-M	1.5×10^{-4}	1.3×10^{-2}	2.5×10^{-3}	1.2×10^{-3}
	B-M	1.3×10^{-3}	2.1×10^{-2}	8.8×10^{-4}	2.8×10^{-3}
	C-M	5.2×10^{-3}	3.1×10^{-3}	3.8×10^{-4}	1.8×10^{-3}
Far-field	A-F	1.5×10^{-4}	1.2×10^{-2}	2.3×10^{-3}	1.2×10^{-4}
	B-F	1.2×10^{-3}	1.8×10^{-2}	8.3×10^{-4}	2.4×10^{-3}
	C-F	4.2×10^{-3}	3.0×10^{-3}	3.6×10^{-4}	1.7×10^{-3}

[a]In this scenario, VOCs do not degrade and are not retarded due to adsorption on aquifer materials. Exposures are calculated from the highest 70-y average concentrations presented in Table 4-4.

[b]See Figure 4-5 for well locations.

5.1.1.3.1. Exposure Via Ingestion of Fruits and Vegetables. We calculated the intake of VOCs in fruits and vegetables from the relation

$$e_{fv} = F_{fv} \times C_w , \quad (5\text{-}13)$$

where

efv = intake of contaminant through fruits and vegetables (mg/kg-d),

Ffv = PEF linking exposure by ingestion of fruits and vegetables to contaminant concentration in irrigation water (L/kg-d), and

Cw = concentration of contaminant in irrigation water (mg/L).

We calculated the concentration of a VOC in fruits and vegetables from the following relation (McKone, 1988):

$$C_b = 0.2 \times K_{sp} \times C_s , \quad (5\text{-}14)$$

where

C_b = concentration of VOC in fresh weight of vegetables (mg/kg),

0.2 = dry mass fraction of fresh vegetables, and

K_{sp} = plant/soil partition factor, which is the ratio of the VOC concentration in biota dry mass per unit concentration in soil C_s (mg/kg).

To estimate the value of C_s, we conservatively assumed that irrigation water containing a dissolved VOC at concentration C_w (mg/L) is frequently added to soil via hand watering or drip irrigation so that the soil-moisture content is maintained. Once incorporated in the soil, VOCs will continue to volatilize to the atmosphere. For the purposes of this analysis, we assumed that 50% of the VOCs will be lost to the atmosphere. Under steady-state conditions, C_s can then be calculated from

$$C_s = 0.5\, C_w K_d , \quad (5\text{-}15)$$

where K_d is the soil/soil water partition coefficient of VOC in soil (L/kg).

We calculated the value of K_{sp} from the regression equation given by Travis and Arms (1988):

$$\log K_{sp} = 1.588 - 0.578 \log K_{ow} , \quad (5\text{-}16)$$

where K_{ow} is the octanol/water partition coefficient. This relation should only be viewed as an approximation because it does not explicitly account for losses of VOCs from plants that would occur through evapotranspiration. Thus, the actual amount of the VOCs in vegetables is likely to be lower than predicted, perhaps not even detectable. Table 5-7 summarizes the input parameters for PCE, TCE, chloroform, and the other VOCs used to calculate the values of C_s and C_b.

McKone (1988) reviewed food-consumption data from a diet survey presented in Yang and Nelson (1986) and estimated mean daily intakes per kilogram of body weight for children (0 to 15 y) as 0.008 kg/kg-d and for adults (15 to 70 y) as 0.0045 kg/kg-d. The time-weighted average daily intake of the two age groups (I_{fv}) is 5.2×10^{-3} kg/kg-d. Combining the above expressions, we obtain

$$F_{fv} = 0.5 \times K_d \times 0.2 \times K_{sp} \times I_{fv} . \quad (5\text{-}17)$$

Table 5-7 lists the values of F_{fv} for the VOCs. Tables 5-8 and 5-9 present the estimated intakes of fruits and vegetables for the best-estimate and health-conservative simulations, respectively, of the transport of VOCs in ground water. The estimated daily intakes are lower than the daily doses predicted for the water-based pathways associated with domestic uses of tap water. In the case of PCE, for example, the maximum predicted intake from the ingestion of fruits and vegetables irrigated with PCE-containing well water under the health-conservative case for well C-M (1.9×10^{-4} mg/kg-d) is a factor of 27 lower than the total exposure to PCE present in tap water (i.e., 5.2×10^{-3} mg/kg-d). In all likelihood, the actual intake of PCE and the other VOCs via fruits and vegetables is considerably lower than our estimates because we used daily intakes of fruits and vegetables that were based on average dietary intakes, not intakes related to home gardens.

5.1.1.3.2. Sprinkler Irrigation of Vineyard. Well 14C2 is used periodically as a source of sprinkler irrigation water for the vineyard located adjacent to East Avenue. Two potential exposure pathways are associated with such irrigation: inhalation of VOCs that have volatilized from sprinklers and the ingestion of grapes or wine derived from the vineyard. The ingestion pathway would be insignificant, compared with the home-garden scenario assessed previously, for several reasons. First, most of the VOCs in sprinkler irrigation water would be volatilized into air, and any subsequent uptake into grapevines would be largely lost through evapotranspiration. Moreover, the grapes are not produced for direct consumption as raisins or table grapes, but for wine production, which involves processing (e.g., dilution, fermentation) that would effectively reduce or eliminate any residual VOCs.

Table 5-7. Input parameters for PCE, TCE, chloroform, and the "other VOCs" in ground water used to calculate the values of F_{fv}.

VOC	K_d (L/kg)	log K_{ow}[a]	K_{sp}	F_{fv} (L/kg-d)
PCE	2.9[b]	3.14	0.59	0.0009
TCE	1.3[b]	2.42	1.5	0.001
Chloroform	0.49[b]	1.97	2.8	0.0007
Other VOCs[c]	0.16[d]	2.13	2.3	0.00019

[a]From Mallon, 1989 (also see Appendix Table Q-4 in Thorpe *et al.*, 1990).

[b]Calculated as the average of four K_d values reported in Mallon, 1989.

[c]Used in the exposure assessment for 1,1-DCE and carbon tetrachloride.

[d]Calculated from a retardation factor of 2. See Table 4-2.

Table 5-8. Estimated intakes of fruits and vegetables irrigated with VOC-contaminated water predicted for the best-estimate scenario.[a]

Far-field well[b,c]	Best-estimate scenario (dose rate in mg/kg-d) PCE	TCE	Chloroform	Other VOCs
A-F	N/A[d]	N/A[d]	1.1×10^{-7}	N/A[d]
B-F[e]	N/A[d]	1.0×10^{-7}	N/A[d]	1.9×10^{-9}
B-F[e]	N/A[d]	8.0×10^{-8}	7.0×10^{-9}	3.8×10^{-9}
C-F	9.0×10^{-9}	N/A[d]	N/A[d]	N/A[d]

[a]Exposures are calculated from estimated maximum 70-y average concentrations presented in Table 4-3 .

[b]See Figure 4-5 for well locations. The far-field wells represent municipal wells in central Livermore.

[c]Under the best-estimate scenario, the predicted VOC concentrations for the far-field monitor wells in Table 4-3 were reduced by a factor of 10 to account for the dilution processes associated with the pumping of municipal wells (see Section 4.3.5.3). The estimated dose rates presented here were calculated with the adjusted concentrations.

[d]Not applicable. Predicted concentrations were below the detection limit of 0.1 ppb.

[e]This observation point had the highest 70-y concentrations for TCE and the "other VOCs." However, the predicted concentrations for the two VOCs occur at different times; thus, exposure estimates are reported separately.

Table 5-9. Estimated intakes of fruits and vegetables irrigated with VOCs in potential monitor wells predicted for the health-conservative scenario.[a]

Far-field well[b]		Health-conservative scenario (dose rate in mg/kg-d) PCE	TCE	Chloroform	Other VOCs
Near-field	A-N	5.6×10^{-6}	2.6×10^{-4}	3.9×10^{-5}	3.4×10^{-6}
	B-N	4.7×10^{-5}	4.7×10^{-4}	1.4×10^{-5}	8.0×10^{-6}
	C-N	2.4×10^{-4}	6.2×10^{-5}	5.7×10^{-6}	4.8×10^{-6}
Mid-field	A-M	5.3×10^{-6}	2.4×10^{-4}	3.4×10^{-5}	3.4×10^{-6}
	B-M	4.7×10^{-5}	3.8×10^{-4}	1.2×10^{-5}	7.6×10^{-6}
	C-M	1.9×10^{-4}	5.8×10^{-5}	5.1×10^{-6}	4.9×10^{-6}
Far-field	A-F	5.5×10^{-6}	2.3×10^{-4}	3.2×10^{-5}	3.4×10^{-6}
	B-F	4.4×10^{-5}	3.4×10^{-4}	1.1×10^{-5}	6.7×10^{-6}
	C-F	1.5×10^{-4}	5.6×10^{-5}	4.8×10^{-6}	4.6×10^{-6}

[a]Exposures are calculated from estimated maximum 70-y average concentrations presented in Table 4-4.

[b]See Figure 4-5 for well locations.

To determine whether the volatilization of VOCs from sprinklers could represent a significant exposure pathway, we carried out a screening calculation of potential inhalation exposures. We calculated the emission rate of a VOC to the atmosphere by multiplying the volumetric flow rate of the well by the predicted concentration of a VOC. For a 378 L/min flow rate (100 gal/min), the maximum emission of PCE from sprinkler water, based on a predicted concentration of 0.012 mg/L in well D-2 (nearest the vineyard; see Fig. 4-5) under the health-conservative scenario, is 0.076 mg/s. If the well actually operates continuously for 4 months of the year, the average emission rate would be 0.025 mg/s, assuming complete volatilization of PCE in sprinkler water.

The annual average ground-level concentration at a distance r from a constant point source Q (Turner, 1982) is

$$\chi = \frac{Q}{Lu\sigma_y(r)}, \tag{5-18}$$

where

χ = annual average ground level concentration (mg/m^3),

Q = annual average source term (mg/s),

L = annual average mixing height (m),

u = annual average wind speed (m/s),

$\sigma_y(r)$ = annual average standard deviation across the plume width (m), and

r = distance from the source (m).

The standard deviation σ_y as a function of distance r in an arbitrarily selected sector from 16 downwind sectors is given by Turner (1982) as

$$\sigma_y(r) = \frac{2\pi r}{16}. \tag{5-19}$$

With conservative values for annual average mixing height and wind speed of 500 m and 1 m/s, respectively, the associated annual average concentration of PCE 100 m downwind from the source would be 1.3×10^{-6} mg/m^3. If the actual annual average values for mixing height and wind speed for the Livermore Valley were used, the resulting concentration would be a factor of about 3 lower. The estimated daily dose resulting from an alveolar inhalation rate of 0.12 m^3/kg-d (Bogen *et al.*, 1988) and 13% metabolism would be 2×10^{-8} mg/kg-d (i.e., $0.12 \times 0.13 \times 1.3 \times 10^{-6}$). Because this particular exposure pathway is insignificant compared to the other pathways we considered, our estimates of health risk associated with predicted concentrations of the VOCs in well waters focuses on the pathways considered earlier (i.e., household uses and garden irrigation).

5.1.1.4. Summary

In this section, we have assessed the water-based exposures associated with the use of water from various receptor wells downgradient from LLNL. In both the best-estimate and health-conservative scenarios, we calculated exposures assuming that an individual uses well water as the sole source of domestic water for a continuous period of 70 y. We also used the highest 70-y average concentration predicted for each well under both scenarios.

Under the health-conservative scenario, we assumed that wells could be drilled immediately downgradient of LLNL, even though residential areas to the west of LLNL are already supplied with municipal water. To be conservative, we further assumed that such wells were completed directly into the zone of contamination and were located in the central portions of the main contaminant plumes. No credit was taken for the in-well dilution that would occur if a well draws water from noncontaminated zones. Furthermore, as discussed in Section 4 of this document, we do not account for VOC degradation and adsorption onto aquifer material.

In the best-estimate case, we focus on the municipal wells in central Livermore as the wells of greatest concern. The maximum predicted concentrations for wells in that area were reduced by a factor of 10 to account for in-well dilution, as discussed in Section 4.3.5.3.

Table 5-10 summarizes the values we used to represent various attributes of different population groups. Tables 5-11 and 5-12 present predicted exposures for the best-estimate and health-conservative scenarios, respectively. Our analyses show that water ingestion, inhalation, and dermal uptake dominate the predicted exposures in either scenario. Consumption of garden vegetables irrigated with contaminated well water contributes little to total exposures.

5.1.2. Potential Public-Health Risks

Our goal in this part of the investigation was to assess health risks associated with the potential VOC exposures presented in Section 5.1.1. Our assessment is divided into two parts. The first part addresses potential cancer risks associated with PCE, TCE, and chloroform, which account for an estimated 91% of the VOCs dissolved in ground water, and carbon tetrachloride, which is used as an indicator compound to assess the carcinogenic potential of other VOCs. The second part addresses the health risks posed by 1,1-DCE, the primary organic compound in the remaining 9% of the VOCs in ground water.

We calculated the potential cancer risks associated with long-term (70-y) exposures to PCE, TCE, chloroform, and carbon tetrachloride by multiplying the previously estimated dose rates by the estimated carcinogenic potency of each compound. The carcinogenic potency is the probability of inducing excess cancer over background risk per unit dose rate, where potency is in units of $(\text{mg of VOC/kg body weight per day})^{-1}$. The carcinogenic potential of a compound is evaluated using a weight-of-evidence approach. This approach takes into consideration information such as the genotoxicity of a compound, results of epidemiological studies, pharmacological data, and, most importantly, the results of lifetime animal bioassays. Potencies for these VOCs are calculated by fitting a dose-response model to the data on tumor incidence from an animal bioassay. The linearized, multistage dose-response model (most frequently applied to tumor-incidence data) predicts a finite increased risk of cancer for any dose rate above zero, with increased low-dose risk linearly proportional to dose rate. This extrapolation is based

Table 5-10. Summary of exposure-related parameters used in the health-risk assessment.

Age group	Body weight (kg)	Body surface area (m^2)	Tap-water intake (L/d)	Breathing rate (L/kg-h)	Garden produce intake (kg/kg-d)
Infants/young children	11.6	0.59	0.44	19	0.008[a]
Older children/teens	38.0	1.2	0.75	19	0.008[a]
Adults	71.8	1.8	1.21	13.5	0.0045

[a]Calculated for children 0 to 15 y.

Table 5-11. Summary of the exposures predicted for the best-estimate exposure scenario.

Well	Adjusted[a] concentration (µg/L or ppb)	Water ingestion (mg/kg-d)	Produce ingestion (mg/kg-d)	Inhalation (mg/kg-d)	Dermal uptake (mg/kg-d)	Totals (mg/kg-d)
Well A-F						
Chloroform	0.15	2.7E–06	1.1E–07	2.4E–06	2.7E–06	7.9E–06
Well B-F[b]						
TCE	0.1	1.8E–06	1.0E–07	1.7E–06	1.9E–06	5.5E–06
Other VOCs	0.01	1.8E–07	1.9E–09	2.4E–07	2.7E–07	6.9E–07
Well B-F[c]						
TCE	0.08	1.4E–06	8.0E–08	1.4E–06	1.5E–06	4.4E–06
Chloroform	0.01	1.8E–07	7.0E–09	1.6E–07	1.8E–07	5.3E–07
Other VOCs	0.02	3.6E–06	3.8E–09	4.8E–07	5.4E–07	1.4E–06
Well C-F						
PCE	0.01	1.8E–07	9.0E–09	3.1E–08	3.5E–08	2.6E–07

[a]Predicted concentrations (from Table 4-3) have been reduced by a factor of 10 to account for the in-well dilution that will occur as a municipal well draws water from both contaminated and uncontaminated zones.

[b]Time period of maximum TCE concentration.

[c]Time period of maximum carbon tetrachloride concentration.

on the assumption that these substances cause cancer in humans at dose rates that are substantially below the doses administered to laboratory animals. A noncarcinogen, in contrast, is not assumed to produce toxic responses in exposed individuals unless a threshold intake is exceeded. Thus, in assessing the health risk of exposure to 1,1-DCE, which we treat as a noncarcinogen, the primary consideration is whether a reference dose rate (RfD) is exceeded.

Table 5-12. Summary of the exposures predicted for the health-conservative exposure scenario in which VOCs do not degrade and are not retarded due to adsorption on aquifer materials. Predicted concentrations are from Table 4-4.

Well	Predicted concentration (µg/L or ppb)	Water ingestion (mg/kg-d)	Produce ingestion (mg/kg-d)	Inhalation (mg/kg-d)	Dermal uptake (mg/kg-d)	Totals (mg/kg-d)
Well A-N						
PCE	6.2	1.1E–04	5.6E–06	1.9E–05	2.2E–05	1.6E–04
TCE	260	4.7E–03	2.6E–04	4.4E–03	4.9E–03	1.4E–02
Chloroform	55	9.9E–04	3.9E–05	8.8E–04	9.9E–04	2.9E–03
Other VOCs	18	3.2E–04	3.4E–06	4.3E–04	4.9E–04	1.2E–03
Well B-N						
PCE	52	9.4E–04	4.7E–05	1.6E–04	1.8E–04	1.3E–03
TCE	470	8.5E–03	4.7E–04	8.0E–03	8.9E–03	2.6E–02
Chloroform	20	3.6E–04	1.4E–05	3.2E–04	3.6E–04	1.1E–03
Other VOCs	42	7.6E–04	8.0E–06	1.0E–03	1.1E–03	2.9E–03
Well C-N						
PCE	270	4.9E–03	2.4E–04	8.4E–04	9.5E–04	6.9E–03
TCE	62	1.1E–03	6.2E–05	1.1E–03	1.2E–03	3.4E–03
Chloroform	8.2	1.5E–04	5.7E–06	1.3E–04	1.5E–04	4.3E–04
Other VOCs	25	4.5E–04	4.8E–06	6.0E–04	6.8E–04	1.7E–03
Well A-M						
PCE	5.9	1.1E–04	5.3E–06	1.8E–05	2.1E–05	1.5E–04
TCE	240	4.3E–03	2.4E–04	4.1E–03	4.6E–03	1.3E–02
Chloroform	49	8.8E–04	3.4E–05	7.8E–04	8.8E–04	2.6E–03
Other VOCs	18	3.2E–04	3.4E–06	4.3E–04	4.9E–04	1.2E–03
Well B-M						
PCE	52	9.4E–04	4.7E–05	1.6E–04	1.8E–04	1.3E–03
TCE	380	6.8E–03	3.8E–04	6.5E–03	7.2E–03	2.1E–02
Chloroform	17	3.1E–04	1.2E–05	2.7E–04	3.1E–04	9.0E–04
Other VOCs	40	7.2E–04	7.6E–06	9.6E–04	1.1E–03	2.8E–03
Well C-M						
PCE	210	3.8E–03	1.9E–04	6.5E–04	7.4E–04	5.4E–03
TCE	58	1.0E–03	5.8E–05	9.9E–04	1.1E–03	3.2E–03
Chloroform	7.3	1.3E–04	5.1E–06	1.2E–04	1.3E–04	3.8E–04
Other VOCs	26	4.7E–04	4.9E–06	6.2E–04	7.0E–04	1.8E–03

Table 5-12. (Continued)

Well	Predicted concentration (µg/L or ppb)	Water ingestion (mg/kg-d)	Produce ingestion (mg/kg-d)	Inhalation (mg/kg-d)	Dermal uptake (mg/kg-d)	Totals (mg/kg-d)
Well A-F						
PCE	6.1	1.1E–04	5.5E–06	1.9E–05	2.1E–05	1.6E–04
TCE	230	4.1E–03	2.3E–04	3.9E–03	4.4E–03	1.3E–02
Chloroform	45	8.1E–04	3.2E–05	7.2E–04	8.1E–04	2.4E–03
Other VOCs	18	3.2E–04	3.4E–06	4.3E–04	4.9E–04	1.2E–03
Well B-F						
PCE	49	8.8E–04	4.4E–05	1.5E–04	1.7E–04	1.2E–03
TCE	340	6.1E–03	3.4E–04	5.8E–03	6.5E–03	1.9E–02
Chloroform	16	2.9E–04	1.1E–05	2.6E–04	2.9E–04	8.4E–04
Other VOCs	35	6.3E–04	6.7E–06	8.4E–04	9.5E–04	2.4E–03
Well C-F						
PCE	170	3.1E–03	1.5E–04	5.3E–04	6.0E–04	4.3E–03
TCE	56	1.0E–03	5.6E–05	9.5E–04	1.1E–03	3.1E–03
Chloroform	6.9	1.2E–04	4.8E–06	1.1E–04	1.2E–04	3.6E–04
Other VOCs	24	4.3E–04	4.6E–06	5.8E–04	6.5E–04	1.7E–03

5.1.2.1. Potential Health Risks Attributable to the Predicted Exposures for PCE, TCE, Chloroform, and Carbon Tetrachloride

We begin our risk assessment with brief reviews of the carcinogenicity of PCE, TCE, chloroform, and carbon tetrachloride, the principal VOCs of concern. Appendix D contains additional information on the genotoxic effects of the compounds and details of animal bioassays. We then present estimates of the cancer risks for the water-based exposures for tap water use in the home and irrigation of home gardens calculated in the previous section.

5.1.2.1.1. Trichloroethylene. TCE has yielded variable results in short-term tests of genotoxicity. Many of the negative or weakly positive results were obtained from bacterial test systems with *Salmonella typhimurium* and *Escherchia coli*. TCE has induced mitotic recombination in *Saccharomyces cerevisiae* and a slight increase in unscheduled DNA synthesis in cultured human cells. There is evidence that suggests TCE can covalently bind to DNA in the presence of metabolic activation.

In animals, TCE has induced malignant liver tumors in male and female B6C3F1 mice [National Cancer Institute (NCI), 1976a; National Toxicology Program (NTP), 1983; Bell *et al.*, 1978], hepatomas in male Swiss mice (Henschler *et al.*, 1980), pulmonary adenocarcinomas in female ICR mice (Fukuda *et al.*, 1983), pulmonary tumors in B6C3F1 and Swiss mice (Maltoni *et al.*, 1986), renal adenomas and carcinomas in F344/N rats (NTP, 1983) and Osborne-Mendel rats (NTP, 1988), and testicular tumors in Marshall and Sprague-Dawley rats (NTP, 1988; Maltoni *et al.*, 1986).

For humans, the currently available epidemiological evidence is inconclusive. No direct relation has been demonstrated between occupational or environmental exposure to TCE and an increased risk of human cancer. Based on its finding of "sufficient evidence" of TCE carcinogenicity to animals and the "inadequate" human evidence, the EPA Carcinogen Assessment Group has classified TCE as a Probable Human Carcinogen (Group B2).

The EPA *Health Assessment Document for Trichloroethylene* (U.S. EPA, 1985a) lists cancer potencies for TCE that range from 5.8×10^{-3} to 1.9×10^{-2} $(mg/kg\text{-}d)^{-1}$. These potencies were derived from data on mouse tumor incidence obtained by the NCI (1976a) and NTP (1983), and are listed in Appendix Table D-1. The EPA (U.S. EPA, 1985a) did not consider data from other bioassays because of concerns regarding data quality (Bell *et al.*, 1978; NTP, 1983, rat data only), relevance (Henschler *et al.*, 1980), or because the studies were not available at the time (Fukuda *et al.*, 1983; Henschler *et al.*, 1984; Maltoni *et al.*, 1986).

An independent review of TCE carcinogenicity was recently completed by Bogen *et al.* (1988). In this analysis, cancer potencies of TCE were calculated by taking into account physiologically based pharmacokinetic (PBPK) models and metabolized dose. A comparison of the methodology used by the EPA (U.S. EPA, 1985a) was also included. Bogen *et al.* calculated cancer potencies for TCE based on 13 sets of tumor-incidence data from studies by the NCI (1976a), NTP (1983), Bell *et al.* (1978), Henschler *et al.* (1980), Fukuda *et al.* (1983), and Maltoni *et al.* (1986). A potency based on each of these data sets was calculated using one of the alternative multistage dose-response models, and using one of two alternative methods for extrapolating toxicologically equipotent doses between species. One method was based on body weight (BW) and the other on surface area (SA). The result was a total of 50 alternative calculated potencies for TCE. Table 5-13 shows the range and median of the 24 of these potency values based on the "linearized," multistage dose-response extrapolation model (U.S. EPA, 1980; Anderson *et al.*, 1983). The potency values that were based on data from the study by Henschler *et al.* (1980) are not included in Table 5-13 because of questions regarding the biological significance of data from this study (Bogen *et al.*, 1988).

The potency values in Table 5-13 reflect 48 one-tailed upper-95%-confidence limits on the slope of the low-dose dose-response curve for TCE-induced carcinogenesis. These values were estimated according to three different versions of the linearized, multistage model for extrapolating dose-response (one time-independent and two time-dependent versions). In addition, two different methods were used for interspecies extrapolation of equipotent dose (referred to above as the BW and SA approaches). Thus, 24 alternative potency values are associated with each of the two approaches to interspecies equipotent-dose extrapolation. These 24 potency values are derived from eight different bioassays of TCE carcinogenicity in male or female mice or rats. Of these values, four were evaluated using each of two closely related tumor endpoints: one involving only malignant tumors of a given type and the other involving those malignant tumors plus histologically related benign tumors. These 24 TCE-related potency values were thus derived from a total of 12 alternative tumor-response data sets, some of which were analyzed using different dose-response extrapolation models.

The large number of alternative potencies, each calculated on a different basis (e.g., tumor types, animal species, and method of dose conversion), makes it difficult to determine which potency to use for the purposes of health-risk assessment. We calculated the median of the TCE

Table 5-13. Range and median of carcinogenic potency values for TCE, PCE, and chloroform. Potencies are based on metabolized doses.

Compound	Number of potency estimates	95% UCL $(mg/kg\text{-}d)^{-1}$ potency[a]			
		Range by SA[b]	Range by BW[c]	Median by SA	Median by BW
TCE[d]	24	0.0020–0.059	0.00034–0.0048	0.017	0.0014
PCE[e]	8	0.095–0.42	0.0073–0.064	0.27	0.023
Chloroform[f]	5	0.0044–0.20	NA[g]	0.028	NA[g]

[a]Potency means the low-dose dose-response slope expressed by an upper-bound, linear, multistage coefficient. At very low doses, risk = potency × dose, according to a multistage (or, with time-to-tumor data as input, a time-dependent multistage) risk prediction model (U.S. EPA, 1980; Anderson *et al.*, 1983; Crump and Howe, 1984). 95% UCL = one-tailed 95% upper confidence limit.

[b]Surface area interspecies dose-extrapolation method. Equivalent doses assumed to be in $mg/kg^{2/3}$.

[c]Body weight interspecies dose-extrapolation method. Equivalent doses assumed to be in mg/kg.

[d]Potency values for TCE are from Bogen *et al.* (1988) based on studies by the NCI (1976a), NTP (1983), Bell *et al.* (1978), Fukuda *et al.* (1983), and Maltoni *et al.* (1986). Together these studies comprise five bioassays including a total of eight species- and sex-specific (12 species-, sex-, and tumor-specific) data sets, which, when up to three different dose-response models per data set were applied, yielded a total of 24 different potency estimates.

[e]Potency values for PCE are from Bogen *et al.* (1987) based on studies by NCI (1977) and NTP (1986). These studies include eight species-, sex-, and tumor-specfic data sets. Application of a dose-response model to each data set yielded the eight separate potency estimates.

[f]Potency values for chloroform are from U.S. EPA (1985b) based on studies by NCI (1976b), Roe *et al.* (1979), and Jorgenson *et al.* (1985). These studies include five species-, sex-, and tumor-specific data sets. Application of a dose-response model to each data set yielded the five different potency estimates.

[g]Potency values for chloroform based on a body-weight interspecies dose-extrapolation method were not provided by the U.S. EPA (1985b).

potencies to serve as a best-estimate for this compound. Moreover, to be conservative, we chose the median potency calculated from a surface-area extrapolation of doses. To evaluate the consequence of using this median value, we compared it to values derived from a composite distribution for all of the potency data sets.

In this technique, the entire uncertainty distribution representing estimation error for the linear parameter in the exponentiated polynomial of a multistage model is generated. The resulting distribution may then be combined with alternative distributions for uncertainty in carcinogenic potency as well as with distributions representing uncertainty and/or interindividual variability in other key components of a modeled risk-generating process. The result is an integrated analysis of uncertainty and interindividual variability in predicted risk (Bogen and Spear, 1987).

To illustrate the impact of uncertainty arising from the combination of parameter-estimation error and lack of knowledge regarding the correct basis for interspecies extrapolation of equipotent dose, we applied the "complete-distribution" approach to characterize uncertainty in the linear coefficient, q_1. This coefficient represents TCE carcinogenic potency at low doses based on implementation of the time-independent multistage (TIM) model of Crump and Watson (1979) described by the EPA (U.S. EPA, 1980) and by Anderson *et al.* (1983). Figure 5-3 shows

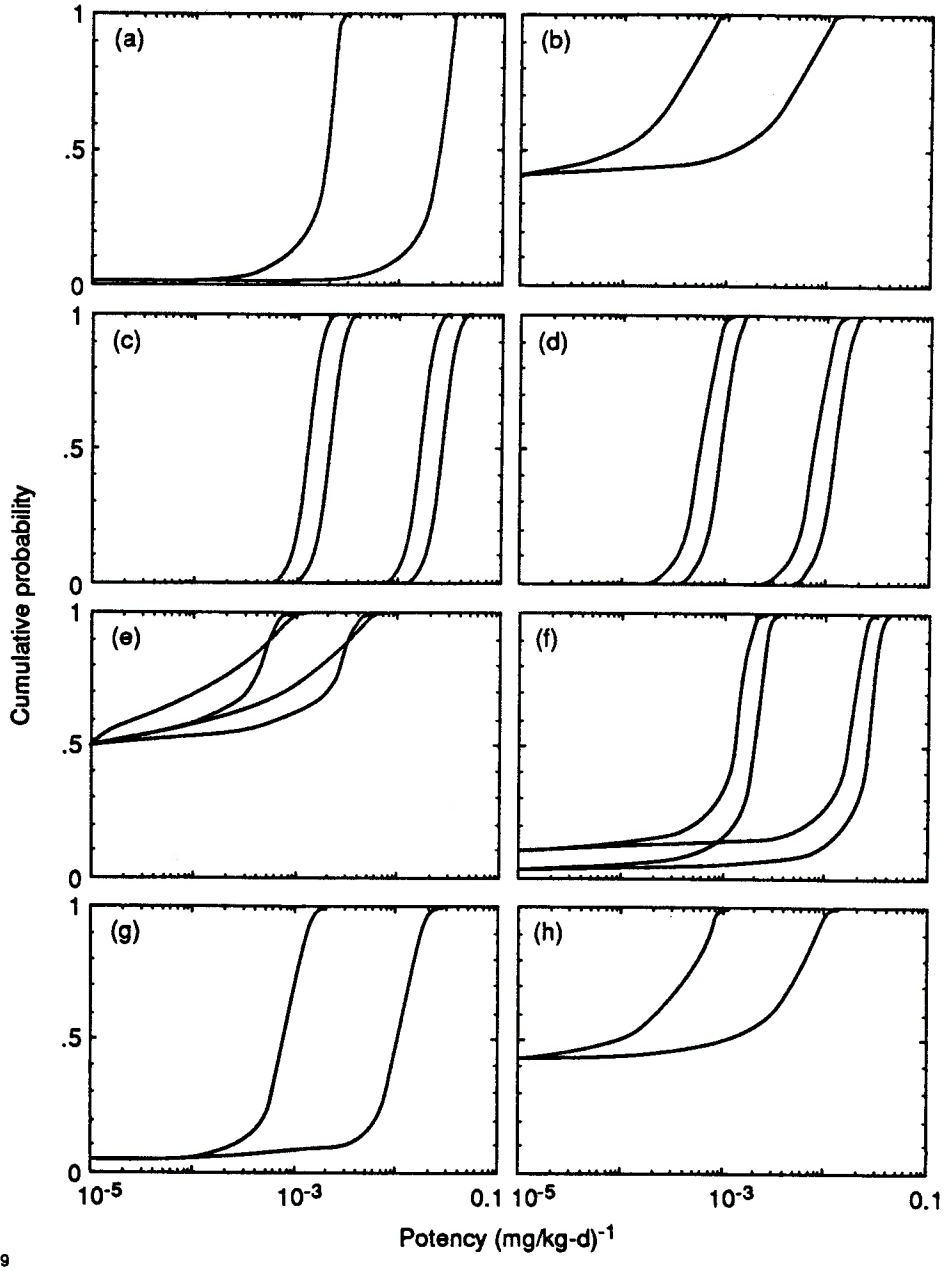

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Figure 5-3. Alternative cumulative probability distribution functions (cdfs) characterizing uncertainty (with regard to parameter-estimation error and lack of knowledge regarding the correct basis for interspecies extrapolation of equipotent dose) in predicted carcinogenicity of low doses of TCE to humans. The eight graphs correspond to potency estimates based on the PBPK-based analysis by Bogen *et al.* (1988) of the following bioassay data. (a) Hepatocellular carcinoma (HCC) in male B6C3F1 mice (NCI, 1976a). (b) HCC in female B6C3F1 mice (NCI, 1976a). (c) HCC (first and third curves from the left) and either HCC or hepatocellular adenoma (HCA) (second and fourth curves from the left) in male B6C3F1 mice (NTP, 1983). (d) HCC (first and third curves from the left) and either HCC or HCA (second and fourth curves from the left) in female B6C3F1 mice (NTP, 1983). (e) Renal tubular-cell (RT) carcinoma (first and third curves from the left, evaluated at P=0.75) and RT adenoma (second and fourth curves from left, evaluated at P=0.75) in male F344/N rats (NTP, 1983). (f) HCC (first and third curves from left) and HCA (second and fourth curves from left) in male B6C3F1 mice (Bell *et al.*, 1978). (g)Lung adenocarcinoma in female ICR mice (Fukuda *et al.*, 1983). (h) Malignant hepatoma in male Swiss mice (Maltoni *et al.*, 1986). Each graph contains one pair [or, in (c) to (f), two pairs] of cdfs whose abcissa values differ (on a log scale) by a bioassay-specific constant.

the resulting 24 cumulative distribution functions (cdfs) for potency associated with each of the eight bioassays of TCE carcinogenicity in rodents. These cdfs are based on the TIM model using both the BW and SA approaches to interspecies dose-equivalence extrapolation. For four of these bioassays, two cdfs shown per bioassay represent the alternative cdfs obtained from applying the BW and SA approaches to interspecies dose-equivalence extrapolation. For the four remaining bioassays, the four cdfs shown for each bioassay represent the alternative cdfs obtained from applying the BW and SA methods to the calculation of potencies. These potencies were derived from tumor-incidence data sets involving either purely malignant tumors or the combination of those malignant tumors plus corresponding, histologically related, benign tumors.

For the purposes of this illustration, a cdf characterizing overall uncertainty (in the restricted context of this analysis) in the linear TIM coefficient q_1 was calculated for TCE from the 24 cdfs shown in Figure 5-3. The calculation was based on the assumption that each of eight bioassays (corresponding to the eight graphs in Fig. 5-3) has equal weight or *a priori* probability as a basis for an estimate of TCE carcinogenic potency in humans. In addition, we assumed that each of the cdfs pertaining to a given bioassay has equal weight or *a priori* probability as an estimate of TCE potency to humans predicted by that bioassay. Thus, the cdfs from graphs a, b, g, and h in Figure 5-3 were each assigned a weight of 1/16. The remaining cdfs in all other graphs were each assigned a weight of 1/32. The resulting composite cdf for q_1, shown in Figure 5-4, is the sum of the latter 24 weighted cdfs. This sum was calculated using a Monte Carlo procedure that generated the cdf of 32,000 potency values randomly sampled from the 24 weighted cdfs. Based on this calculated composite cdf, the median carcinogenic potency of TCE to humans [1.6×10^{-3} $(mg/kg\text{-}d)^{-1}$] is lower, by a factor of approximately 35, than the highest upper-bound potency value for TCE listed in Table 5-13. It is about a factor of 10 lower than the median of the upper-95%-confidence-limit potencies. Based on this analysis, we conclude that the median of SA-derived potencies is not likely to understate the risk of exposures to TCE.

5.1.2.1.2. Tetrachloroethylene. Depending on the organism, test system, and purity of the sample, PCE has yielded both positive and negative results in tests of genotoxicity. However, the majority of results from bacterial assays of mutagenesis have been negative. PCE has induced mitotic recombination and gene conversion in *S. cerevisiae*. There is some evidence that PCE can induce unscheduled DNA synthesis in isolated hepatocytes. In animals, PCE has induced malignant liver tumors in male and female B6C3F1 mice (NCI, 1977; NTP, 1986) and mononuclear cell leukemia in rats (NTP, 1986). The epidemiological evidence for PCE is limited, and the relationship between exposure to PCE and an elevated risk of cancer is equivocal.

In 1985, the EPA concluded that the evidence for the carcinogenicity of PCE in animals was "limited" and that the epidemiological data were "inconclusive." Consequently, PCE was placed by the EPA in Group C, a Possible Human Carcinogen. Since this evaluation, the final report of the NTP (1986) inhalation bioassay of PCE was made public. In this document, the NTP concluded that, under the conditions of the study, there was "clear evidence of carcinogenicity" of PCE for male F344/N rats, "some evidence of carcinogenicity" of PCE for female F344/N rats, and "clear evidence of carcinogenicity" for both sexes of B6C3F1 mice. Based on the additional evidence from the NTP bioassay, the Carcinogen Assessment Group of the EPA determined that there was "sufficient" evidence for the carcinogenicity of PCE in animals but

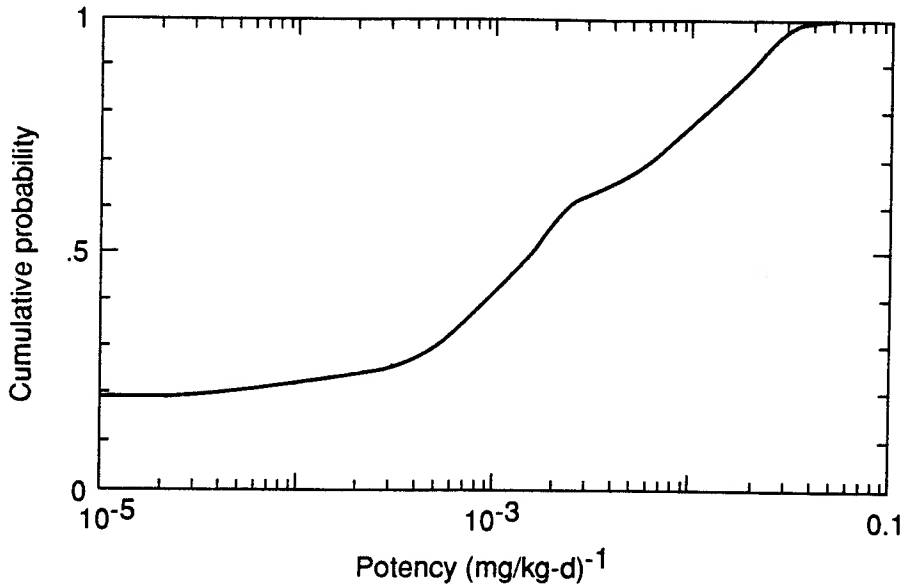

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Figure 5-4. Composite cumulative probability distribution function (cdf) characterizing uncertainty (with regard to parameter-estimation error and lack of knowledge regarding the correct basis for interspecies extrapolation of equipotent dose) in predicted carcinogenicity of low doses of TCE to humans. This composite cdf was calculated from the information shown in Figure 5-3 under the assumption that each of the cdfs within any given graph is equally likely and that the eight sets of cdfs comprising the eight graphs are equally likely.

that the evidence of its carcinogenicity to humans remained "inconclusive." Accordingly, PCE was placed in Group B2, as a Probable Human Carcinogen. However, the classification of PCE is currently under review by the EPA, and a final determination has not been made (U.S. EPA, 1990).

The EPA *Health Assessment Document for Tetrachloroethylene* (U.S. EPA, 1985c) lists cancer potencies for PCE that range from 5.1×10^{-2} to 1.6×10^{-1} $(mg/kg\text{-}d)^{-1}$. The potencies were derived from data on mouse tumor incidence obtained by the NCI (1977). The EPA (U.S. EPA, 1985c) did not consider tumor-incidence data from the NTP (1986) study of PCE because it had not been peer-reviewed at the time of publication. The EPA subsequently published an addendum to the 1985 document (U.S. EPA, 1986b) that listed cancer potencies for PCE based on mouse- and rat-tumor-incidence data obtained by the NTP (1986) (see Appendix Table D-8). These potencies range from 1.0×10^{-3} to 3.3×10^{-3} $(mg/kg\text{-}d)^{-1}$.

Bogen *et al.* (1987) calculated cancer potencies for PCE based on eight sets of tumor-incidence data from studies of the NCI (1977) and NTP (1986). Potencies based on the steady-state pharmacokinetic approach (described in Bogen *et al.*, 1987) resulted in the range and median values listed in Table 5-13. As documentation to support an MCL for PCE in California drinking water, the California DHS published cancer potency values for PCE that range from 4.1×10^{-3} to 8.5×10^{-2} $(mg/kg\text{-}d)^{-1}$ (DHS, 1988).

As noted in our discussion of TCE, the number of alternative potency values for PCE and the different methods used to calculate each make it difficult to choose a single value as the best-estimate of PCE carcinogenic potential. Consequently, we selected the median potency of 0.27

$(mg/kg\text{-}d)^{-1}$ calculated from a surface-area extrapolation of metabolized dose for use in our health-risk assessment (see Table 5-13).

5.1.2.1.3. Chloroform. The results of short-term testing of chloroform for mutagenicity in bacteria have been negative, with one exception. This reportedly positive study was compromised by poorly documented results (Agustin and Lim-Sylianco, 1978). There is also limited evidence that chloroform may be weakly clastogenic and that it may induce sperm-head abnormalities. Data are inconclusive with respect to the ability of chloroform to bind to or damage DNA.

The carcinogenicity of chloroform in animals has been evaluated in seven strains of mice, two strains of rats, and in beagle dogs. Chloroform induced a significantly increased incidence of:

- Renal tumors in male rats when administered in drinking water (Jorgenson *et al.*, 1985).
- Hepatic adenofibrosis in male and female rats when administered in drinking water (Tumasonis *et al.*, 1985 and 1987).
- Hepatocellular carcinomas in male and female mice when administered by gavage in corn oil (NCI, 1976b).
- Renal tumors in male mice of the ICI strain (i.e., a strain of mice sharing a common origin in the Imperial Chemical Industries Ltd. colony at Alderley Park, MacClesfield, Cheshire, United Kingdom) when administered orally (Roe *et al.*, 1979).
- Hepatomas in strain A mice when administered orally (Eschenbrenner and Miller, 1945).

On the basis of the positive results of Eschenbrenner and Miller (1945), NCI (1976b), and Roe *et al.* (1979), the IARC (1982) concluded that there was "sufficient" evidence that chloroform is a carcinogen in animals. The IARC also noted that there was inadequate evidence to evaluate chloroform's carcinogenicity to humans. Similarly, the EPA (U.S. EPA, 1985b, 1990) concluded that there was "sufficient" evidence that chloroform is carcinogenic to animals. The EPA considered the epidemiological evidence on chloroform to be "inadequate" to evaluate its carcinogenicity to humans. Consequently, chloroform was placed in Group B2, a Probable Human Carcinogen.

The EPA *Health Assessment Document for Chloroform* (U.S. EPA, 1985b) lists cancer potencies for chloroform that range from 4.4×10^{-3} to 2.0×10^{-1} $(mg/kg\text{-}d)^{-1}$ (Table 5-13). These potencies were derived from data on mouse tumor incidence and male rat tumor incidence obtained by the NCI (1976b) from data on mouse tumor incidence reported by Roe *et al.* (1979) and from the combined incidence of renal adenomas and adenocarcinomas in male rats (Jorgenson *et al.*, 1985). These data are summarized in Appendix Tables D-9 and D-10. The EPA (1985b) did not consider the data of Reuber (1979) in its calculation of potencies, apparently because his conclusions were considered to be the result of a difference of opinion between pathologists. Data on tumor incidence by Tumasonis *et al.* (1985, 1987) were not available to the EPA at the time of publication.

The IRIS database for chloroform (U.S. EPA, 1990) lists an oral cancer potency of 6.1×10^{-3} $(mg/kg\text{-}d)^{-1}$ and an inhalation cancer potency of 8.1×10^{-2} $(mg/kg\text{-}d)^{-1}$. The oral potency was calculated from the rat kidney tumor data of Jorgenson *et al.* (1985). However, the data set used in this instance included *all* kidney tumors, not only the histologically related adenomas and

adenocarcinomas used in the earlier EPA analysis (U.S. EPA, 1985b). The inhalation cancer potency was based on the incidence of hepatocellular carcinoma in female mice in the NCI (1976b) bioassay of chloroform. For our health-risk assessment of chloroform, we again selected the median cancer potency, 0.028 $(mg/kg\text{-}d)^{-1}$, calculated from a surface-area extrapolation of dose from the range of available values (Table 5-13).

5.1.2.1.4. Health Risks of Water-Based Exposures to PCE, TCE, Chloroform, and Carbon Tetrachloride. Tables 5-14 and 5-15 summarize the estimated cancer risks associated with maximum 70-y exposures (via water ingestion, inhalation of VOCs volatilized from showers, and dermal absorption) calculated for both the best-estimate and health-conservative exposure cases for PCE, TCE, and chloroform. In addition, we present the estimated cancer risks for carbon tetrachloride, which we use as an indicator compound to assess the cancer risk for other VOCs. Under the best-estimate exposure scenario (Table 5-14), the greatest cancer risk of 2×10^{-7} is associated with the far-field well A-F, which is in the path of the plume containing an elevated concentration of chloroform. Under the health-conservative exposure scenario, the total risks are on the order of 10^{-3} to 10^{-4} for all wells. The highest predicted risk (2×10^{-3}) is for a potential monitor well (C-N) that is 75 m from the LLNL site. The uniformity of risk is due primarily to the assumptions regarding the lack of degradation and retardation of the VOCs. These assumptions result in peak 70-y concentrations that are not reduced significantly by the distance traveled.

5.1.2.2. Potential Health Risks Attributable to 1,1-DCE in Ground Water

Of the various minor organic compounds detected in ground water at the LLNL site (an estimated 9% of the total mass of VOCs in ground water), 1,1-DCE is the most important. It constitutes nearly 60% of the minor organic constituents. In this subsection, we assess the potential health hazard posed by this compound.

5.1.2.2.1. RfD Values for 1,1-DCE Compared with Predicted Exposures. Both the EPA (U.S. EPA, 1990) and the California DHS (1988) have derived drinking water standards for 1,1-DCE based on the results of a chronic toxicity study conducted by Quast *et al.* (1983). In this study, male and female Sprague-Dawley rats were given 1,1-DCE in drinking water over a 2-y period. The authors calculated that males received 7, 10, or 20 mg/kg-d, and females, 9, 14, or 30 mg/kg-d of 1,1-DCE. The oral RfD originally calculated by the EPA (U.S. EPA, 1985d) and used to calculate the Federal MCL was based on the determination that Quast *et al.* (1983) defined a lowest observed adverse-effect level (LOAEL) for 1,1-DCE of 10 mg/kg-d. However, the DHS (1988) and, later the EPA (U.S. EPA, 1990) noted that Quast *et al.* (1983) observed midzonal fatty degeneration of the liver in female rats at all dose levels. Consequently, both agencies determined that the data of Quast *et al.* actually define a chronic LOAEL of 9 mg/kg-d for 1,1-DCE in female rats. The current EPA oral RfD for 1,1-DCE of 0.009 mg/kg-d was calculated by dividing the LOAEL of 9 mg/kg-d by an uncertainty factor of 1,000.

Both the Federal and State MCLs for 1,1-DCE were calculated by including an additional uncertainty factor of 10 to account for the conflicting results of carcinogenicity studies. (See App. D for a discussion of the carcinogenicity of 1,1-DCE.) We note, however, that the MCL derived by the DHS (6 μg/L) and the EPA (7 μg/L) (see Table 3-5) differ slightly because the EPA has not revised the Federal MCL following the revision of the RfD (DHS, 1988; U.S. EPA, 1990).

Table 5-14. Summary of predicted incremental cancer risks for the best-estimate exposure scenario.

Well[a]	Predicted concentration[b] (μg/L)	Total exposure[c] (mg/kg-d)	Cancer potency [(mg/kg-d)$^{-1}$]		Risk[d]
Well A-F					
Chloroform	0.15	7.9E–06	0.028		2E–07
				Total =	2E–07
Well B-F[e]					
TCE	0.1	5.5E–06	0.017		9E–08
Other VOCs	0.01	6.9E–07			
Carbon tetrachloride[f]	0.0044	3.0E–07	0.13[g]		4E–08
				Total =	1E–07
Well B-F[h]					
TCE	0.08	4.4E–06	0.017		7E–08
Chloroform	0.01	5.3E–07	0.028		1E–08
Other VOCs	0.02	1.4E–06			
Carbon tetrachloride[f]	0.009	6.2E–07	0.13[g]		8E–08
				Total =	2E–07
Well C-F					
PCE	0.01	2.6E–07	0.27		7E–08
				Total =	7E–08

[a]See Figure 4-5 for well locations.

[b]The predicted concentrations from Table 4-3 have been reduced by a factor of 10 to account for the dilution that would occur as the municipal wells draw uncontaminated ground water from different vertical and horizontal zones associated with the screened intervals of the wells.

[c]Exposures are for all pathways.

[d]Values are rounded to one significant figure.

[e]Time period of maximum TCE concentration.

[f]The predicted 70-y concentration for the other VOCs in ground water is multiplied by 0.44 to obtain the estimated concentration for carbon tetrachloride, which we use as the indicator compound for the VOCs in a portion of the ground water.

[g]Cancer potency is based on applied dose, not metabolized dose.

[h]Time period of maximum carbon tetrachloride concentration.

Table 5-16 shows the predicted exposures for the best-estimate and health-conservative transport scenarios compared with the RfD value. Using the RfD of 0.009 mg/kg-d (U.S. EPA, 1990), we find that none of the predicted exposures exceed the RfD (i.e., ratios of exposure to RfD are less than 1). However, if we apply an additional uncertainty factor of 10 to the RfD to

Table 5-15. Summary of predicted incremental cancer risks for the health-conservative exposure scenario.

Well[a]	Predicted concentration[b] (mg/L)	Total exposure[c] (mg/kg-d)	Cancer potency [(mg/kg-d)$^{-1}$]		Risk[d]
Well A-N					
PCE	0.0062	1.6E–04	0.27		4E–05
TCE	0.26	1.4E–02	0.017		2E–04
Chloroform	0.055	2.9E–03	0.028		8E–05
Carbon tetrachloride	0.0079	5.3E–04	0.13		7E–05
				Total =	4E–04
Well B-N					
PCE	0.052	1.3E–03	0.27		4E–04
TCE	0.47	2.6E–02	0.017		4E–04
Chloroform	0.02	1.1E–03	0.028		3E–05
Carbon tetrachloride	0.018	1.3E–03	0.13		2E–04
				Total =	1E–03
Well C-N					
PCE	0.27	6.9E–03	0.27		2E–03
TCE	0.062	3.4E–03	0.017		6E–05
Chloroform	0.0082	4.3E–04	0.028		1E–05
Carbon tetrachloride	0.011	7.5E–04	0.13		8E–05
				Total =	2E–03
Well A-M					
PCE	0.0059	1.5E–04	0.27		4E–05
TCE	0.24	1.3E–02	0.017		2E–04
Chloroform	0.049	2.6E–03	0.028		7E–05
Carbon tetrachloride	0.0079	5.3E–04	0.13		7E–05
				Total =	4E–04
Well B-M					
PCE	0.052	1.3E–03	0.27		4E–04
TCE	0.38	2.1E–02	0.017		4E–04
Chloroform	0.017	9.0E–04	0.028		3E–05
Carbon tetrachloride	0.018	1.2E–03	0.13		2E–04
				Total =	1E–03

Table 5-15. (Continued)

Well[a]	Predicted concentration[b] (mg/L)	Total exposure[c] (mg/kg-d)	Cancer potency [(mg/kg-d)$^{-1}$]		Risk[d]
Well C-M					
PCE	0.21	5.4E–03	0.27		1E–03
TCE	0.058	3.2E–03	0.017		5E–05
Chloroform	0.0073	3.8E–04	0.028		1E–05
Carbon tetrachloride	0.011	7.5E–04	0.13		8E–05
				Total =	1E–03
Well A-F					
PCE	0.0061	1.6E–04	0.27		4E–05
TCE	0.23	1.3E–02	0.017		2E–04
Chloroform	0.045	2.4E–03	0.028		7E–05
Carbon tetrachloride	0.0079	5.3E–04	0.13		7E–05
				Total =	4E–04
Well B-F					
PCE	0.049	1.2E–03	0.27		3E–04
TCE	0.34	1.9E–02	0.017		3E–04
Chloroform	0.016	8.4E–04	0.028		2E–05
Carbon tetrachloride	0.015	1.0E–03	0.13		1E–04
				Total =	7E–04
Well C-F					
PCE	0.17	4.3E–03	0.27		1E–03
TCE	0.056	3.1E–03	0.017		5E–05
Chloroform	0.0069	3.6E–04	0.028		1E–05
Carbon tetrachloride	0.01	7.5E–04	0.13		1E–04
				Total =	1E–03

[a]See Figure 4-5 for well locations.

[b]The predicted 70-y concentrations for the other VOCs in Table 4-4 have been multiplied by 0.44 to obtain estimates of the concentrations of carbon tetrachloride in the various wells.

[c]Exposures for all pathways.

[d]Values rounded to one significant figure.

account for the contradictory data on carcinogenicity, then nearly all of the health-conservative exposures exceed this adjusted RfD (ratios range from 0.74 to 1.8). Given the magnitude of the ratios (i.e., less than 2) and the conservative exposure assumptions, it is unlikely, even in this case, that 1,1-DCE constitutes a health threat.

Table 5-16. Comparisons of the RfD value for 1,1-DCE, developed by the U.S. EPA (1990), with the best-estimate and health-conservative exposures. All exposures for 1,1-DCE are calculated by multiplying the maximum exposures for "other VOCs" by 0.56, the fraction of 1,1-DCE in this category.

Well group/monitor well[a]		Best-estimate case			Health-conservative case		
		Exposure [mg/kg-d ($\times 10^{-6}$)]	RfD [mg/kg-d ($\times 10^{-3}$)]	Ratio [exposure/RfD ($\times 10^{-4}$)]	Exposure [mg/kg-d ($\times 10^{-3}$)]	RfD [mg/kg-d ($\times 10^{-3}$)]	Ratio (exposure/RfD)
Near-field	A-N	N/A[b]	N/A[b]	N/A[b]	0.67	9	0.074
	B-N	N/A[b]	N/A[b]	N/A[b]	1.6	9	0.18
	C-N	N/A[b]	N/A[b]	N/A[b]	0.95	9	0.11
Mid-field	A-M	N/A[b]	N/A[b]	N/A[b]	0.67	9	0.074
	B-M	N/A[b]	N/A[b]	N/A[b]	1.6	9	0.18
	C-M	N/A[b]	N/A[b]	N/A[b]	1.0	9	0.11
Far-field	A-F	ND[c]	9	—	0.67	9	0.074
	B-F	0.78	9	0.87	1.3	9	0.14
	C-F	ND[c]	9	—	0.95	9	0.11

[a]See Figure 4-5 for well locations. In the best-estimate case, no wells are assumed to be complete in just the contaminated permeable sediments directly in the paths of VOC plumes.

[b]Not applicable. Under the best-estimate scenario, near- and mid-field wells are considered improbable.

[c]Concentrations below detection level of 0.1 ppb.

5.1.2.3. Conclusions

Our primary goal was to assess the potential health risks associated with exposures to ground waters contaminated with VOCs originating at the LLNL site. As a means of estimating the range of possible exposures, we defined two transport scenarios, one representing a set of best-estimate values for the transport and fate parameters, and the other representing what we have termed health-conservative values that yield higher concentrations of VOCs in ground water. In addition, we located three sets of potential monitor wells at locations where the highest concentrations of VOCs are expected to occur west of the LLNL site. Three receptor wells were co-located with existing domestic and irrigation wells to the south of East Avenue. The predicted 70-y VOC concentrations in those wells were not adjusted to reflect the dilution that would occur in wells that draw water from different aquifer zones.

At present, no domestic wells west of the LLNL site in the near- and mid-field zones are in the paths of the principal plumes of VOCs. Our analyses indicate that it is unlikely that new wells will be drilled in those areas because the subdivisions located there are currently supplied by city water, and the cost of drilling a private well for inferior water (ground water that is high in total dissolved solids) would be prohibitive. Therefore, the predicted exposures to VOCs and

associated health risks for the wells in those areas have a low probability of actually occurring. Existing wells south of East Avenue that are used for domestic or agricultural purposes are free of contamination; hence, there are no exposures to VOCs. To ensure that exposures do not occur, wells near the existing VOC plumes are monitored on a regular basis. Our simulations of the transport and fate of VOCs for wells D-1 and D-3 indicate that existing domestic wells south of East Avenue are not likely to become contaminated, even assuming that the plumes move in a westerly direction.

Exposures to VOCs in well water were shown to be dominated by three pathways: water ingestion, inhalation of VOCs that have volatilized during showers, and dermal uptake while bathing. The incremental cancer risks for the best-estimate scenario for exposure to VOCs in municipal wells near central Livermore were on the order of 10^{-7} to 10^{-8}. Under the health-conservative scenario, the lifetime cancer risks for the combined exposures to the VOCs were on the order of 10^{-3} to 10^{-4} for all wells because the 70-y average concentrations were not reduced by transformation processes. The exposure period associated with the maximum 70-y concentration of the VOCs in the far-field monitor wells was 110 y in the future, compared with 270 y for the best-estimate case.

Our analysis of the predicted water-based exposures to 1,1-DCE, representing the remaining VOCs in the plumes of ground water contamination, showed that the RfD (safe intake rate) for 1,1-DCE developed by the EPA (U.S. EPA, 1990) was not exceeded under either the best-estimate or health-conservative transport scenarios. However, if an additional safety factor of 10 is applied to the RfD to account for conflicting information on its carcinogenicity, then the RfD would be exceeded by a factor of up to 2 for a near-field well in the health-conservative transport scenario.

Finally, we have computed exposures and associated risks for the two scenarios using EPA methodologies and data. The results of those analyses are presented in Appendix E. In general, the results of those analyses are in reasonable agreement with those presented in this section. For example, predicted cancer risks for the best-estimate exposure scenario range from 10^{-6} to 10^{-8} for the far-field wells, representing municipal wells in downtown Livermore. For the health-conservative exposure case, lifetime cancer risks for all wells are approximately 10^{-3}—similar to our results. We also calculated hazard index values for the VOCs of concern as a means of determining whether chronic exposures to those compounds had the potential for causing noncarcinogenic effects. For the best-estimate exposure scenario, the hazard index values were well below 1. For the health-conservative case, some wells attained a hazard index rating of one, which suggests that if the substances acted in a cumulative fashion (even though each VOC was below an applicable RfD value) there could possibly be systemic effects.

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List of Acronyms

1,1-DCA	1,1-dichloroethane
1,1-DCE	1,1-dichloroethylene
1,1,1-TCA	1,1,1-trichloroethane
ADI	acceptable daily intake
AL	action level (for drinking water)
ALPE	Arroyo Las Positas East
ALPW	Arroyo Las Positas West
aos	adult-onsite exposure
ASE	Arroyo Seco East
ASN	Arroyo Seco North
ASS	Arroyo Seco South
AVR	alveolar ventilation rate
BAAQMD	Bay Area Air Quality Management District
BETX	sum of benzene, ethylbenzene, toluene, and xylene isomers
BPHA	Baseline Public Health Assessment
BR	breathing rate
BW	body weight
CAL	contract analytical laboratory
CDB	Central Drainage Basin
cdf	cumulative distribution function
CEC	cation exchange capacity
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CPF	carcinogenic potency factor
CWSC	California Water Service Company
DCA	dichloroethane
DCG	derived concentration guide
DHS	California Department of Health Services
DNA	deoxyribonucleic acid
DOE	U.S. Department of Energy
EAF	Environmental Attenuation Factor
ENU	ethylnitrosourea
EPA	U.S. Environmental Protection Agency
FS	*CERCLA Feasibility Study for the LLNL Livermore Site*
GC/MS	gas chromatography/mass spectrometry
GM	geometric mean
GSD	geometric standard deviation
ha	hectare
HEAST	Health Effects Assessment Summary Tables
HGPRT	hypoxanthine-guanine phosphororibosyl transferase
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System (compiled by EPA)
kV	kilovolts
LLNL	Lawrence Livermore National Laboratory
LOAEL	lowest observed adverse-effect level
LOD	limit of detection
MCL	maximum contaminant level
NCI	National Cancer Institute
NOAEL	no observed adverse-effect level
NPL	National Priorities List
NTP	National Toxicology Program
PBPK	physiologically based pharmacokinetic (model)

PCB	polychlorinated biphenyl
PCE	tetrachloroethylene, also tetrachloroethene and perchloroethylene
pCi/L	picocuries per liter
PDF	pathway dose factor
PEF	pathway-exposure factor
RfD	reference dose
RI	*CERCLA Remedial Investigations Report for the LLNL Livermore Site*
RI/FS	Remedial Investigation/Feasibility Study
RWQCB	California Regional Water Quality Control Board
SA	surface area
SAP	surface soil sample from Arroyo Las Positas
SARA	Superfund Amendments and Reauthorization Act
SAS	surface soil sample from Arroyo Seco
SBAS	South Bay Aqueduct System
SNL	Sandia National Laboratories
SSD	storm-drain sample
SSS	surficial soil sample
STLC	soluble threshold limit concentration
TCA	trichloroacetic acid
TCE	trichloroethylene
TCL	target compound list
TDL	total designated level
TDS	total dissolved solids
TFHC	total fuel hydrocarbons
TIM	time-independent multistage (model)
TOC	total organic carbon
TSP	total suspended particles
TTLC	total threshold limit concentration
UCL	upper confidence limit
VOC	volatile organic compound
WET	waste extraction test

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Appendix A

Development of Pathway-Exposure Factors and Supporting Calculations for Deriving Maximum, Total-Equivalent Lifetime Exposure Values

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Development of Pathway-Exposure Factors and Supporting Calculations for Deriving Maximum, Total-Equivalent Lifetime Exposure Values

J. I. Daniels, D. W. Layton, and R. T. Cederwall

In this appendix, we derive the pathway-exposure factors (PEFs) used to estimate the potential maximum, total-equivalent lifetime soil-based exposures (E_{max}s) to the organic and inorganic chemicals reported to be at concentrations above the limit of detection (LOD) and above background levels, respectively, in the soils and sediments on and near the LLNL site.

The PEFs incorporate information on human physiology, human behavior patterns, and environmental transport into a term that translates a unit concentration for a chemical in soil into daily exposure, in mg/kg-d, for a given route of exposure, i (where i may be ingestion, inhalation, or dermal absorption). Accordingly, we use the PEF for an applicable route of exposure together with the maximum concentration of a chemical of concern in soil to estimate for a population at risk (i.e., adults onsite at the LLNL site or the public offsite) the potential maximum, equivalent lifetime soil-based exposure ($e_{i\text{-max}}$) for that compound and exposure pathway. The E_{max} value for an organic or inorganic compound and exposed population is equal to the sum of the $e_{i\text{-max}}$ values for the compound and exposed population.

As explained in Section 3, adults onsite at the LLNL site may be exposed to chemicals in soil by

- Ingestion of contaminated soil.
- Inhalation of either contaminated soil particles resuspended into air or organic chemicals volatilized continuously to the atmosphere from an area where the soil is contaminated to a significant depth.
- Dermal absorption of organic chemicals from soil particles deposited on exposed skin surfaces.

The public offsite, however, may be exposed to organic chemicals in soil by only one pathway—inhalation of organic chemicals volatilized continuously to the atmosphere and transported offsite from an area where the soil is contaminated to a significant depth. However, the public might be exposed to inorganic chemicals in soils offsite by ingestion and dermal absorption pathways.

The E_{max} values calculated in the manner just described are compared to either verified reference oral doses (RfD) for noncarcinogenic substances or are correlated to a lifetime cancer risk for carcinogenic compounds. Soil containing substance with an E_{max} value greater than the RfD or resulting in a cancer risk exceeding a target range of 10^{-4} to 10^{-6} (see U.S. EPA, 1990) is a candidate for more detailed assessment.

Soil-Based Pathways, Concentrations in Environmental Media, and Exposure Factors

The procedure for deriving PEFs that appears in this appendix was adapted from a recent report by McKone (1988) concerning methods for estimating multipathway exposure to environmental contaminants. As discussed in Section 3, for a given exposure pathway, a PEF translates the concentration of an organic chemical in soil or air into an equivalent, lifetime soil-based exposure (in units of mg/kg-d) for the individuals of a population over a given period of time (e.g., lifetime for the public or a time-weighted, uninterrupted period of employment for adults onsite). We then use the following equation to calculate a maximum value for each equivalent, lifetime soil-based exposure applicable to a population at risk ($e_{i\text{-max}}$) as

$$e_{i\text{-max}}(\text{population at risk}) = C_{\max} \times F_i , \qquad \text{(A-1)}$$

where F_i is a PEF for pathway i and $C_{\max}$ is the maximum recorded concentration of a contaminant in soil or air.

There are four pathways for which $C_{\max}$ must be determined:

- Ingestion of soil particles.
- Dermal absorption.
- Inhalation of soil particles.
- Inhalation of chemicals volatilized from soil to the atmosphere.

For ingestion of soil particles (i = 1), as well as for dermal absorption of organic chemicals from soil particles deposited on exposed skin surfaces (i = 3), $C_{\max}$ in Eq. (A-1) is equal to the maximum concentration of an organic chemical reported in soil, denoted $C_{s\text{-max}}$. For inhalation outdoors of soil particles (i = 2a), $C_{\max}$ in Eq. (A-1) is equal to the maximum concentration of an organic chemical on soil particles in air, denoted $C_{p\text{-max}}$. For inhalation of chemicals volatilized to the atmosphere from soil (i = 2b), $C_{\max}$ in Eq. (A-1) is equal to the maximum concentration of an organic chemical detected in air that is attributed to exhalation from soil, $C_{a\text{-max}}$. The $C_{s\text{-max}}$ values for the organic chemicals at concentrations above the LOD in soil are obtained directly from the soil/sediment sampling studies. However, both the $C_{p\text{-max}}$ and $C_{a\text{-max}}$ values for these chemicals must be estimated. The derivation of estimates for these parameters is described next.

Derivation of $C_{p\text{-max}}$ and $C_{a\text{-max}}$ Values

Calculation of $C_{p\text{-max}}$

An estimate of $C_{p\text{-max}}$ for an organic chemical in soil is directly related to the concentration of the chemical in the uppermost layer of the soil. This is because contaminated soil particles near the surface are always subject to resuspension in air and subsequent inhalation. The estimate of $C_{p\text{-max}}$ for an organic chemical is obtained by multiplying the value for $C_{s\text{-max}}$ for that chemical, which is in units of mg/kg(soil), by an upper limit for the concentration of total suspended particles (TSP) in urban air, which is in units of kg(particles, including soil)/m^3(air). Bidleman (1988) estimated the average urban TSP concentration to be 9.8×10^{-8} kg/m^3 by using the value of Whitby (1978) for average total volume of urban aerosols [7.0×10^{-11}

cm^3/cm^3(air)] and by assuming a particle density of 1.4 g/cm^3. Bidleman (1988) noted that the calculated value is somewhat higher than the mean TSP reported by Shah *et al.* (1986) for 46 cities in 1975 [7.9×10^{-8} kg/m^3(air)]. However, Bidleman (1988) also explained that the specific surface area of the *average* background aerosol is only 3.6 m^2/g. This value for surface area is about one-third the 11-m^2/g value calculated for *urban* air based on the average values of Whitby (1978) for total surface area [1.1×10^{-5} cm^2/cm^3(air)] and total volume [7.0×10^{-11} cm^3/cm^3(air)] for urban aerosols, and an assumed particle density of 1.4 g/cm^3. Therefore, we consider the value for the average urban TSP concentration calculated by Bidleman (1988) to be an upper limit for the concentration of soil in urban air. Accordingly, we use this value to calculate values of $C_{p\text{-}max}$.

Calculation of $C_{a\text{-}max}$

A soil-based estimate of $C_{a\text{-}max}$ is derived for an organic chemical only if that chemical contaminates soil both at the surface and deep in the vadose zone. This is because only these chemicals volatilize continuously from the vadose zone into the near surface soil and then into the atmosphere where they are transported downwind to populations onsite and offsite. Thus, the derivation of a $C_{a\text{-}max}$ value for these organic chemicals is related to their volatilization rate from the soil and the distance downwind of the population at risk of inhaling these volatilized chemicals.

For screening, we consider only the concentrations of TCE in the surface and deep vadose zone of soil near Building 518. This is because TCE is the predominant VOC at this location, and these concentrations of TCE could support continuous volatilization of this chemical into the atmosphere. Furthermore, the surface and deep vadose zone of soil near Building 518 have higher levels of TCE and other VOCs than any other soils onsite. Because subsequent atmospheric transport of this compound offsite from this location is of concern, we derive an estimated value for $C_{a\text{-}max}$(public) for TCE at the closest site boundary. We also derive an estimated value of $C_{a\text{-}max}$(aos) for TCE for employees working at the location of this soil contamination.

As VOCs, such as TCE, volatilize, the concentration in the soil diminishes. Therefore, the exhalation rate from soil will slowly diminish as well. Exposures based on estimated $C_{a\text{-}max}$ values are, therefore, conservative because such diminishing rates of exhalation of VOCs from soil are not addressed.

The volatilization of a VOC, such as TCE, from near-surface soils to the atmosphere results in a potential inhalation-exposure pathway. To determine the significance of this exposure pathway for the public at the site boundary closest to the contaminated soil near Building 518 and for adults onsite in the contaminated area near Building 518, we calculated a maximum concentration for TCE in air at these two locations resulting from soil exhalation and atmospheric transport.

The maximum average volatilization rate of an organic chemical through the soil surface to the atmosphere over a period of t days can be estimated from the following equation (derived from Dragun, 1988):

$$G = C_T [D_E/(3.1416 \times t)]^{1/2}, \qquad \text{(A-2)}$$

where

G = maximum volatilization rate (mg/m^2-d),

C_T = total initial concentration of contaminant in soil (mg/m^3),

D_E = effective diffusion coefficient of a contaminant in soil (m^2/d), and

t = time (d).

The value of D_E is estimated from the relationship

$$D_E = \{[D_A K_h (P_{sa}^{10/3}/P_T^2)] + [D_W(W_c^{10/3}/P_T^2)]\} \times (B K_d + W_c + P_{sa} K_h)^{-1}, \quad \text{(A-3)}$$

where

D_A = diffusion coefficient of the chemical in air (m^2/d),

K_h = Henry's Law constant (dimensionless),

P_{sa} = soil air content (m^3/m^3),

P_T = total soil porosity (m^3/m^3),

D_W = diffusion coefficient of the chemical in water (m^2/d),

W_c = soil water content (m3/m^3),

B = soil bulk density (kg/m^3), and

K_d = adsorption coefficient of chemical in soil (m^3/kg).

To calculate the value of D_E for TCE in soils in the Building 518 area, we used the following inputs (mainly from Spencer *et al.*, 1988):

- $D_A = 0.432$ m^2/d,
- $D_W = 4.32 \times 10^{-5}$ m^2/d,
- $P_{sa} = 0.1$,
- $P_T = 0.41$,
- $W_c = 0.31$, and
- $B = 1{,}500$ kg/m^3.

The Henry's Law constant (K_h) is equal to 0.265, which is based on a measured value from Gossett (1987) (0.00632 atm-m^3/mol at 17.5°C). The K_d for TCE is 1.3 (or 1.3×10^{-3} m^3/kg),

the average of four values for aquifer material from four boreholes [see Appendix Table Q-5 of the RI report (Thorpe *et al.*, 1990)]. The estimate of D_E is then

$$D_E = \{[0.432 \times 0.265 \times (0.1^{10/3}/0.41^2)] + [4.32 \times 10^{-5} \times (0.31^{10/3}/0.41^2)]\}$$

$$\times (1500 \times 1.3 \times 10^{-3} + 0.31 + 0.1 \times 0.265)^{-1}$$

$$= 1.3 \times 10^{-4} \text{ m}^2/\text{d} .$$

The initial concentration of TCE in soil, C_T, can be calculated from the concentration of TCE on soil particles (as determined by a soil extraction) as

$$C_T = R_S C_S , \quad \text{(A-4)}$$

where

$$R_S = B + W_c/K_d + P_{sa} \times K_h/K_d . \quad \text{(A-5)}$$

The data from the Carpenter (1984) soil survey of the Building 518 area indicates that the near-surface (0.9 to 1.5 m) concentrations of TCE ranged from about 10 to 3,000 µg/kg (the maximum concentration of TCE at depth was 5.7 mg/kg). For this screening calculation, we assume that the soil is uniformly contaminated at a concentration, C_S, of 1 mg/kg. The value of R_S is

$$R_S = 1{,}500 + [0.31/(1.3 \times 10^{-3})] + [0.10 \times 0.265/(1.3 \times 10^{-4})]$$

$$= 1{,}759 \text{ kg/m}^3 .$$

C_T is, therefore, equal to 1,759 mg/m^3.

The value of G (average over 365 d) can now be calculated as

$$G = 5{,}844\left[\frac{1.3 \times 10^{-4}}{3.1416 \times 365}\right]^{1/2}$$

$$= 0.6 \text{ mg/m}^2\text{-d} .$$

Now, if we assume that a 100-m^2 area adjacent to Building 518 (an area which is greater than investigations would suggest) is uniformly contaminated with TCE at a level of 1 mg/kg, then the flux is 60 mg/100 m^2-d.

We used a point-source Gaussian diffusion model to determine the maximum concentration of TCE at the site perimeter. The dispersion equation chosen predicts the ground-level concentration of a contaminant along the plume centerline.

The annual average ground-level concentration at a distance, r, from a constant point source, Q, is (from Turner, 1982)

$$C_{\text{a-max(public)}} = \frac{Q}{Lus_y(r)} , \quad \text{(A-6)}$$

where

$C_{a\text{-}max}$(public) = annual average ground level concentration (mg/m^3),

Q = annual average source term (mg/s),

L = annual average mixing height (m),

u = annual average wind speed (m/s),

s_y (r) = annual average standard deviation across the plume width (m), and

r = distance from the source (m).

The standard deviation, s_y, as a function of distance, r, in an arbitrarily selected sector from 16 downwind sectors is given by Turner (1982) as

$$s_y(r) = \frac{2\pi r}{16}. \qquad \text{(A-7)}$$

The distance from Building 518 to the site boundary is about 380 m, so the value of s_y (r) is 149 m. With conservative values for annual average wind speed and mixing height of 1 m/s and 500 m, the value of $C_{a\text{-}max}$(public)/Q from Eq. (A-6) is 1.3×10^{-5} s/m^3. So, with an exhalation rate of 6.9×10^{-4} mg/s, the downwind annual average concentration, $C_{a\text{-}max}$(public), becomes 9×10^{-9} mg/m^3. If the actual annual average values for mixing height and wind speed for the Livermore Valley were used, the resulting concentration would be about a factor of three lower.

We now calculate the maximum concentration of TCE exhaled from soil into the air for adults onsite (aos) in the contaminated area near Building 518. For screening purposes, we calculate this value, $C_{a\text{-}max}$(aos), using the following conservative assumptions and using Eq (A-6) to reflect the parameters associated with these assumptions. First, we assume that adults onsite are exposed at the downwind edge of the contaminated area because this maximizes the amount of exhaled TCE to which they are exposed. Second, we assume that the TCE released at the surface of the contaminated area is mixed sufficiently by turbulence to create a uniform concentration from the surface to a height of 1.5 m (presumed to be the height at which inhalation takes place). Third, we assume a wind speed of 1 m/s, which minimizes the dilution volume of air into which the TCE is exhaled from the soil.

We now reduce the mixing height, L, in Eq. (A-6) to the height at which inhalation takes place (1.5 m) and also use the width of the 100-m^2 contaminated area (assumed to be 10 m on a side) for the value of s_y. The substitution of these values into Eq. (A-6) results in a value for $C_{a\text{-}max}$(aos) equal to 4.6×10^{-5} mg/m^3. This value of $C_{a\text{-}max}$(aos) is consistent with that obtained using the formula for dispersion of material from an area source described by Hanna *et al.* (1982).

Calculation of PEFs and Corresponding $e_{i\text{-}max}$ Values

As discussed in Section 3, for the purpose of screening calculations we make the following conservative assumptions to determine the value of a PEF (F_i) for use in Eq. (A-1) either for adults onsite (aos) or for the public. First, we assume that the exposure period for the public

offsite for any exposure pathway of concern is equal to a 70-y lifetime. Second, we assume that the exposure period for aos for any pathway of concern is equal to 50 y of continuous exposure outdoors and that the aos have 70-y lifespans. Finally, we compute PEFs for the public, F_i(public), for a 70-y lifetime based on component PEFs for children, f_i(child), and adults, f_i(adult), and we compute PEFs for adults onsite based on the component PEF for an adult, f_i(adult). As stated in Section 3, the equations for calculating F_i(public) and F_i(aos) are

$$F_i(\text{public}) = [(15/70) \times f_i(\text{child})] + [55/70 \times f_i(\text{adult})] , \quad \text{(A-8)}$$

and

$$F_i(\text{aos}) = (50/70) \times f_i(\text{adult}) . \quad \text{(A-9)}$$

In Eq. (A-8), the factors 15/70 and 55/70 are estimates of the fraction of a 70-y lifespan an individual in the population spends as a child and an adult, respectively. In Eq. (A-9), the factor 50/70 represents a conservative estimate of the fraction of a 70-y lifespan an individual employed at the LLNL site spends working outdoors.

For the reasons discussed in Section 3, the only potential exposure pathway for the public for an organic chemical present in soil at the Livermore site is by inhalation following continuous emission to the atmosphere and subsequent transport of the chemical offsite from a source area. Consequently, for organic chemicals, Eq. (A-8) is used to calculate only one PEF for the public, F_{2b}(public), and as mentioned already, only for TCE. However, for adults onsite, PEFs are developed for ingestion of soil particles, F_1(aos); for inhalation of soil particles outdoors, F_{2a}(aos); and for dermal absorption of chemicals from soil particles deposited on exposed skin surfaces, F_3(aos). A PEF term for TCE addressing inhalation of TCE exhaled from the soil adjacent to Building 518 is also calculated for adults onsite outdoors in that area, F_{2b}(aos).

The specific F_i values for use in Eqs. (A-8) and (A-9) are developed next. Then, the maximum, total-equivalent lifetime exposure for the public offsite, E_{max}(public), and for adults onsite, E_{max}(aos), are calculated by summing the respective $e_{i\text{-max}}$(population) values.

Ingestion of Soil Particles

A review of empirical data on human soil ingestion by LaGoy (1987) indicates that the estimated average intake of soil for adults who engage in outdoor activities or exhibit frequent hand to mouth activities is 50 mg/d. Dividing this intake rate by the average weight for an adult of 66.5 kg (derived from data on weights of male and female adults in ICRP, 1975), and multiplying that product by a factor of 10^{-6}, which relates milligrams of soil to kilograms of soil, yields a component PEF of 7.5×10^{-7} mg/kg-d per mg/kg for ingestion of soil, f_1. Substituting this value of f_1 for the term f_i in Eq. (A-9) results in a value for F_1(aos) equal to 5.4×10^{-7} mg/kg-d per mg/kg.

Appendix Tables A-1 and A-2 contain the $e_{1\text{-max}}$(aos) values for each of the 20 organic and 13 inorganic chemicals identified as being of potential concern in soils at the LLNL site (see Section 3 and Appendix C). Also included in Appendix Tables A-1 and A-2 are the corresponding values for $C_{s\text{-max}}$ and for F_1(aos).

Inhalation of Soil Particles

The component PEF for inhalation of soil particles (f_2) is calculated for a 50-y employment period using an adaptation of the mathematical expression developed for this purpose by McKone (1988):

$$f_{2a} = \{(A)\times[(IW_a \times IQ_{wa}) + (IH_a \times IQ_{ha}) + (SW_a \times SQ_{wa}) + (SH_a \times SQ_{ha})] \times (BR/BW)_a\}$$

$$\{(R) \times [(IW_r \times IQ_{wr}) + (IH_r \times IQ_{hr}) + (SW_r \times SQ_{wr}) + (SH_r \times SQ_{hr})] \times (BR/BW)_{r,}\} \quad \text{(A-10)}$$

where

A = total active hours/calendar day (16),

IW_a = fraction of total active hours spent indoors at work (0/16),

IQ_{wa} = proportion of particulate matter indoors at work compared to that outdoors at work as reported by Hawley (1985) (0.75),

IH_a = fraction of total active hours spent indoors at home (6/16),

IQ_{ha} = proportion of *contaminated* particulate matter indoors at home relative to that outdoors at home (assumed to be zero),

SW_a = fraction of total active hours spent outdoors at work (8/16),

SQ_{wa} = proportion of outdoor particulate matter considered to be contaminated, (1.0 $mg/m^3/mg/m^3$),

SH_a = fraction of total active hours spent outdoors at home (2/16),

SQ_{ha} = proportion of outdoor particulate matter at home considered to be contaminated (zero),

$(BR/BW)a$ = reference arithmetic-mean breathing rate per unit body weight for adults while active (derived from ICRP, 1975) (0.018 m3/kg-h),

R = total resting hours/calendar day (8),

IW_r = fraction of total resting hours spent indoors at work (0/8).

IQ_{wr} = proportion of particulate matter indoors at work compared to that outdoors at work as reported by Hawley (1985) (0.75),

IH_r = fraction of total resting hours spent indoors at home (7/8),

IQ_{hr} = proportion of *contaminated* particulate matter indoors at home compared to that outdoors at home (assumed to be zero),

SW_r = fraction of total resting hours spent outdoors at work (1/8),

SQ_{wr} = proportion of outdoor particulate matter considered to be contaminated (1.0 $mg/m^3/mg/m^3$),

SH_r = fraction of total resting hours spent outdoors at home (2/16),

SQ_{hr} = proportion of outdoor particulate matter at home considered to be contaminated (zero), and

$(BR/BW)_r$ = reference arithmetic-mean breathing rate per unit body weight for adults while resting (derived from ICRP, 1975) (0.006 m^3/kg-h).

Substituting the numerical values just noted for the corresponding terms in Eq. (A-10) yields the following expression:

$$f_{2a} = [(16 \text{ total active h/d}) \times (8 \text{ active work h/16 total active h}) \times (1.0 \text{ mg/m}^3/\text{mg/m}^3) \times (0.018 \text{ m}^3/\text{kg-h})] + [(8 \text{ total resting h/d}) \times (1 \text{ resting work h/8 total resting h}) \times (1.0 \text{ mg/m}^3/\text{mg/m}^3) \times (0.006 \text{ m}^3/\text{kg-h})]$$

$$= 0.15 \text{ mg/kg-d per mg/m}^3 . \quad \text{(A-11)}$$

Substituting the value just calculated for f_{2a} for f_i in Eq. (A-9) yields a value for F_{2a}(aos) equivalent to 0.11 mg/kg-d per mg/m^3. Appendix Tables A-3 and A-4, respectively, contain the values for $e_{2a\text{-}max}$(aos) for each of the 20 organic and 13 inorganic chemicals identified as being of potential concern in soils at the LLNL site [see Section 3 and Appendices O and P of the RI report (Thorpe *et al.*, 1990)]. Also presented in Appendix Tables A-3 and A-4 are the corresponding values for $C_{s\text{-}max}$, $C_{p\text{-}max}$, and F_{2a}.

Inhalation of Chemicals Volatilized from the Soil to the Atmosphere

The component PEFs, f_{2b}(child) and f_{2b}(adult), for inhalation by the public offsite of TCE volatilized to the atmosphere from soils at the LLNL site are obtained from the following mathematical expressions that were adapted from the work of McKone (1988) and Bogen *et al.* (1988):

$$f_{2b}(\text{child}) = [(A) \times (BR/BW)_{ca} \times MF \times AVF] + [(R) \times (BR/BW)_{cr} \times MF \times AVF] , \quad \text{(A-12)}$$

and

$$f_{2b}(\text{adult}) = [(A) \times (BR/BW)_{aa} \times MF \times AVF] + [(R) \times (BR/BW)_{ar} \times MF \times AVF] , \quad \text{(A-13)}$$

where

A = total active hours/calendar day (16 h/d),

$(BR/BW)_{ca}$ = reference arithmetic-mean breathing rate (BR) per unit body weight (BW) for a child while active (0.029 m^3/kg-h) (derived from data in ICRP, 1975),

MF = fraction of TCE in alveolar air that is metabolized (0.72),

AVF = factor for converting BR to the alveolar ventilation rate (AVR), assumed to be equal to the ratio between the AVR for adults (353.5 L/h) and the rate BR for adults (1,200 L/h) (derived from data in ICRP, 1975) (0.30),

R = total resting hours/calendar day (8 h/d),

$(BR/BW)_{cr}$ = reference arithmetic-mean BR per unit BW for a child while resting (0.011 m^3/kg-h) (derived from data in ICRP, 1975),

$(BR/BW)_{aa}$ = reference arithmetic-mean BR per unit BW for an adult while active (0.018 m^3/kg-h) (derived from data in ICRP, 1975), and

$(BR/BW)_{ar}$ = reference arithmetic-mean BR per unit BW for an adult while resting (0.006 m^3/kg-h) (derived from data in ICRP, 1975).

Total respiration is converted to an AVR because gases are exchanged almost entirely in the alveolar space of the lungs.

Substituting the numerical values stated above for the terms in Eqs. (A-12) and (A-13) yields

$$f_{2b}(\text{child}) = [(16 \text{ total active h/d}) \times (0.029 \text{ m}^3\text{/kg-h}) \times (0.72) \times (0.30)]$$
$$+ [(8 \text{ total resting h/d}) \times (0.011 \text{ m}^3\text{/kg-h}) \times (0.72) \times (0.30)]$$
$$= 0.119 \text{ m}^3\text{/kg-d} \quad \text{(A-14)}$$

and

$$f_{2b}(\text{adult}) = [(16 \text{ total active h/d}) \times (0.018 \text{ m}^3\text{/kg-h}) \times (0.72) \times (0.30)]$$
$$+ [(8 \text{ total resting h/d}) \times (0.006 \text{ m}^3\text{/kg-h}) \times (0.72) \times (0.30)]$$
$$= 0.072 \text{ m}^3\text{/kg-d}. \quad \text{(A-15)}$$

The component PEFs derived in Eqs. (A-14) and (A-15) are used in Eq. (A-8) to determine $F_{2b\text{-max}}$(public) for TCE, and the component PEF derived in Eq. (A-15) is used in Eq. (A-9) to determine $F_{2b\text{-max}}$(aos) for TCE. The value for $F_{2b\text{-max}}$(public) for TCE obtained using Table A-8 is 0.082 mg/kg-d per mg/m^3 or m^3/kg-d and the value for $F_{2b\text{-max}}$(aos) for TCE obtained using Eq. (A-9) is 0.052 mg/kg-d per mg/m^3 or m^3/kg-d. Next, these values for $F_{2b\text{-max}}$(public) and $F_{2b\text{-max}}$(aos) for TCE are substituted into Eq. (A-1), along with the corresponding values for $C_{a\text{-max}}$(public) (9×10^{-9} mg/m^3) and $C_{a\text{-max}}$(aos) (4.6×10^{-5} mg/m^3), to derive values for $e_{2b\text{-max}}$(public) and $e_{2b\text{-max}}$(aos) for TCE. The resulting values for $e_{2b\text{-max}}$(public) and $e_{2b\text{-max}}$(aos) for TCE are 7.4×10^{-10} mg/kg-d and 2.4×10^{-6} mg/kg-d.

Dermal Absorption of Chemicals from Soil Particles

Dermal absorption of contaminants from soil occurs through the accumulation of contaminated soil on skin. The amount of soil that accumulates on human skin depends on a number of factors such as age, type of soil, exposed surface area, and soil conditions. These factors vary greatly, making the estimation of soil dermal absorption a relatively uncertain

process. To calculate a PEF for dermal absorption from soil, we define the exposure for this pathway in terms of the amount of soil contaminant that passes from the soil matrix on the skin into the underlying tissue. For this screening analysis, we adopted the following assumptions of Hawley (1985) regarding this pathway:

- The absorption rates for pure compounds on the skin surface are on the order of 12% per day for adults.
- The duration of dermal absorption of chemicals from soil is on the order of 12 h/d.
- The percentage of compounds absorbed from a soil matrix is on the order of 15% of that for pure compounds.

We assume that the heads and upper extremities of adults are the portions of the body that accumulate soil particles. According to the ICRP (1975), these components comprise 26% of the surface area of an adult. The arithmetic-mean weight-specific surface area for both male and female adults combined is 0.026 m^2/kg. This figure is calculated by dividing the average surface area for males and females by the average mass for males and females presented in Table 5-2.

Lepow *et al.* (1975) measured the concentration of soil on the hands of children and found an average of 0.005 kg/m^2. Roels *et al.* (1980) measured the amounts of lead on the hands of children compared to that in soil. Their work indicates that soil concentrations on the extremities of children are on the order of 0.05 kg/m^2. Based on these measurements, and the assumption that adults who work outdoors have the same soil concentration on their extremities as children, we assume that adults have soil concentrations on their extremities of 0.03 kg/m^2. Combining the information above gives the following expression for PEFs for soil dermal-absorption:

$$f_3 = PB \times WSSA \times PSM \times DAR \times TDA \times SCE , \qquad \text{(A-16)}$$

where

PB = proportion of body accumulating soil (0.26),

$WSSA$ = average weight-specific surface area (0.026 m^2/kg),

PSM = proportion of compound absorbed from soil matrix (0.15 mg/kg per mg/kg),

DAR = daily absorption rate of compound from soil on skin surface (0.12/d),

TDA = time of dermal absorption over the course of a day (12h/24h), and

SCE = soil concentration on the extremities of an adult (0.03 kg/m^2).

Substituting the numerical values above for the terms in Eq. (A-16) results in a value for f_3 equal to 1.8×10^{-6} mg/kg-d per mg/kg. Replacing f_i in Eq. (A-9) with the value of f_3 results in a value for F_3(aos) equal to 1.3×10^{-6} mg/kg-d per mg/kg. Inserting the value for F_3(aos) that was just determined and the maximum concentration, $C_{s\text{-}max}$, for each of the 20 organic chemicals detected in soil on the LLNL site into Eq. (A-1) yields $e_{3\text{-}max}$(aos) values for each of the 20 organic chemicals. Appendix Tables A-5 and A-6, respectively, contain the maximum concentration data, F_3(aos) values, and $e_{3\text{-}max}$(aos) values for each of the 20 organic and 13

inorganic chemicals identified as being of concern in soils at the LLNL site (see Section 3 and Appendix C).

Calculation of E_{max} Values for Adults Onsite and for the Public

For easy reference, each of the previously determined PEF values (F_i), applicable to chemicals of concern detected in soil on the LLNL site, and the corresponding parameters and units for $C_{i\text{-}max}$ are presented in Appendix Table A-7. The maximum, total-equivalent lifetime exposure for adults onsite, E_{max}(aos), for a specific chemical is determined by summing each of the $e_{i\text{-}max}$(aos) values for that chemical. Similarly, the E_{max}(public) value for an organic chemical is also determined by summing each of the $e_{i\text{-}max}$(public) values for that chemical. However, the value for E_{max}(public) is equal to the value for $e_{2b\text{-}max}$(public), because volatilization to the atmosphere of any VOC is the only pathway by which the public might be exposed. Accordingly, the value for E_{max}(public) is equal to 7.4×10^{-10} mg/kg-d. Appendix Tables A-8 and A-9, respectively, present both the $e_{i\text{-}max}$ and E_{max} values for adults onsite for each of the 20 organic and 13 inorganic chemicals identified as being of possible concern in soil at the LLNL site. As explained previously, for screening purposes the values for $e_{2b\text{-}max}$ for adults onsite (and for the public offsite) were only determined for TCE.

The maximum soil concentrations reported for inorganic chemicals in the arroyos near the LLNL site do not exceed background (see Appendix C). Therefore, relevant multipathway exposure factors and corresponding exposure rates were not derived for the public offsite.

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Appendix Table A-1. Total equivalent lifetime soil-ingestion exposure of adults onsite (aos) $e^{org}_{1\text{-}max}$(aos) for a 50-y period for each of the 20 organic chemicals in soil at the LLNL site with a frequency of detection (>LOD) that is greater than or equal to 5%.

Organic chemical	Location[a]	[$C^{org}_{s\text{-}max}$] Maximum concentration in soil (mg/kg)	[F_1] PEF for ingestion of soil (mg/kg-d per mg/kg)	[$e^{org}_{1\text{-}max}$] Ingestion exposure for adults onsite (mg/kg-d)
Acetone	SSS-009	8.0E+00	5.4E–07	4.3E–06
Aroclor (PCB) 1254	SSD-008	1.3E+00	5.4E–07	7.0E–07
Benzene	ALPW	1.1E–02	5.4E–07	5.9E–09
Chloroform	ASS	9.5E–03	5.4E–07	5.1E–09
1,2-dichlorobenzene	ASN	8.7E–03	5.4E–07	4.7E–09
1,2-dichloroethylene (total 1,2-DCE)	ALPE	1.6E–03	5.4E–07	8.6E–10
Ethylbenzene	ALPW	5.2E–03	5.4E–07	2.8E–09
Fluoranthene	SSS–009	1.8E+00	5.4E–07	9.7E–07
Methyl acetate	SSD-006	2.0E+00	5.4E–07	1.1E–06
Methylene chloride (dichloromethane)	161-2	5.0E–02	5.4E–07	2.7E–08
Methyl ethyl ketone	612-S2, 612-S3	4.0E–01	5.4E–07	2.2E–07
Phenanthrene	SSS-009	1.0E+00	5.4E–07	5.4E–07
Pyrene	SSS-009	1.3E+00	5.4E–07	7.0E–07
Tetrachloroethylene (PCE)	518-1	3.7E–01	5.4E–07	2.0E–07
Toluene	ALPE	8.3E–03	5.4E–07	4.5E–09
1,1,1-trichloroethane (1,1,1-TCA)	513-1	1.4E–01	5.4E–07	7.6E–08
Trichloroethylene (TCE)	518-2	3.0E+00	5.4E–07	1.6E–06
Trichlorofluoromethane (Freon 11)	SSD-013, SSD-014	3.0E–01	5.4E–07	1.6E–07
Trichlorotrifluoroethane (Freon 113)	513-1	1.6E–01	5.4E–07	8.6E–08
Xylene isomers (total)	SSD-006, SSD-010, SSD-013, SSD-014	2.0E–01	5.4E–07	1.1E–07

[a]See Figures 3-12 and 3-13 for sampling locations. As noted in Section 3, the locations of samples SSS-009 and SSD-008 have been remediated.

Appendix Table A-2. Total equivalent lifetime soil-ingestion exposure of adults onsite, $e^{inorg}_{1\text{-}max}$(aos), for a 50-y period for each of the inorganic chemicals in soil at the LLNL site with a maximum concentration (TTLC[a]) distinct from background levels (as discussed in Appendix C).

Inorganic chemical	Location[b]	[$C^{inorg}_{s\text{-}max}$] Maximum soil concentration at LLNL (mg/kg)	[F_1] PEF for ingestion of soil (mg/kg-d per mg/kg)	[$e^{inorg}_{1\text{-}max}$] Ingestion exposure for adults onsite (mg/kg-d)
Antimony	SSS-009	13	5.4E–07	7.0E–06
Arsenic	SSS-003	14	5.4E–07	7.6E–06
Beryllium	SSD-009	4	5.4E–07	2.2E–06
Cadmium	SSS-009	23	5.4E–07	1.2E–05
Chromium (total)	SSS-009	1,500	5.4E–07	8.1E–04
Cobalt	ALPE	22	5.4E–07	1.2E–05
Copper	SSS-009	530	5.4E–07	2.9E–04
Lead	SSD-009	320	5.4E–07	1.7E–04
Mercury	SSD-009	20	5.4E–07	1.1E–05
Molybdenum	SSS-009	16	5.4E–07	8.6E–06
Nickel	SSS-009	670	5.4E–07	3.6E–04
Silver	SSS-009	7.4	5.4E–07	4.0E–06
Zinc	SSD-009	750	5.4E–07	4.1E–04

[a]The total threshold limit concentration (TTLC) is determined by nitric acid digestion (see DHS, 1984).

[b]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in Section 3, the locations of samples SSS-009 and SSD-009 have been remediated.

Appendix Table A-3. Total equivalent lifetime soil-particle-inhalation exposure of adults onsite, $e^{org}_{2a\text{-}max}$(aos), for a 50-y period for each of the 20 organic chemicals in soil at the LLNL site with a frequency of detection (>LOD) that is greater than or equal to 5%.

Organic chemical	Location[a]	$[C^{org}_{s\text{-}max}]$ Maximum concentration in soil (mg/kg)	$[C^{org}_{p\text{-}max}]$ Maximum concentration on suspended soil particles (mg/m^3)	$[F_{2a}]$ PEF for inhalation of soil particles (mg/kg-d per mg/m^3)	$[e^{org}_{2a\text{-}max}]$ Particle inhalation exposure for adults onsite (mg/kg-d)
Acetone	SSS-009	8.0E+00	7.8E–07	1.1E–01	8.6E–08
Aroclor (PCB) 1254	SSD-008	1.3E+00	1.3E–07	1.1E–01	1.4E–08
Benzene	ALPW	1.1E–02	1.1E–09	1.1E–01	1.2E–10
Chloroform	ASS	9.5E–03	9.3E–10	1.1E–01	1.0E–10
1,2-dichlorobenzene	ASN	8.7E–03	8.5E–10	1.1E–01	9.4E–11
1,2-dichloroethylene (total 1,2-DCE)	ALPE	1.6E–03	1.6E–10	1.1E–01	1.7E–11
Ethylbenzene	ALPW	5.2E–03	5.1E–10	1.1E–01	5.6E–11
Fluoranthene	SSS-009	1.8E+00	1.8E–07	1.1E–01	1.9E–08
Methyl acetate	SSD-006	2.0E+00	2.0E–07	1.1E–01	2.2E–08
Methylene chloride (dichloromethane)	161-2	5.0E–02	4.9E–09	1.1E–01	5.4E–10
Methyl ethyl ketone	612-S2, 612-S3	4.0E–01	3.9E–08	1.1E–01	4.3E–09
Phenanthrene	SSS-009	1.0E+00	9.8E–08	1.1E–01	1.1E–08
Pyrene	SSS-009	1.3E+00	1.3E–07	1.1E–01	1.4E–08
Tetrachloroethylene (PCE)	518-1	3.7E–01	3.6E–08	1.1E–01	4.0E–09
Toluene	ALPE	8.3E–03	8.1E–10	1.1E–01	8.9E–11
1,1,1-trichloroethane (1,1,1-TCA)	513-1	1.4E–01	1.4E–08	1.1E–01	1.5E–09
Trichloroethylene (TCE)	518-2	3.0E+00	2.9E–07	1.1E–01	3.2E–08
Trichlorofluoromethane (Freon 11)	SSD-013, SSD-014	3.0E–01	2.9E–08	1.1E–01	3.2E–09
Trichlorotrifluoroethane (Freon 113)	513-1	1.6E–01	1.6E–08	1.1E–01	1.7E–09
Xylene isomers (total)	SSD-006, SSD-010, SSD-013, SSD-014	2.0E–01	2.0E–08	1.1E–01	2.2E–09

[a]See Figures 3-12 and 3-13 for sampling locations. As noted in Section 3, the locations of samples SSS-009 and SSD-008 have been remediated.

Appendix Table A-4. Total equivalent lifetime soil-particle-inhalation exposure of adults onsite, $e^{inorg}_{2a\text{-}max}$(aos), for a 50-y period for each of the inorganic chemicals in soil at the LLNL site with a maximum concentration (TTLC[a]) distinct from background levels (as discussed in Appendix C).

Inorganic chemical	Location[b]	[$C^{inorg}_{s\text{-}max}$] Maximum soil concentration at LLNL (mg/kg)	[$C^{inorg}_{p\text{-}max}$] Maximum concentration on suspended soil particles (mg/m^3)	[F_{2a}] PEF for inhalation of soil particles (mg/kg-d per mg/m^3)	[$e^{inorg}_{2a\text{-}max}$] Particle inhalation exposure (aos) (mg/kg-d)
Antimony	SSS-009	13	1.3E–06	1.1E–01	1.4E–07
Arsenic	SSS-003	14	1.4E–06	1.1E–01	1.5E–07
Beryllium	SSD-009	4	3.9E–07	1.1E–01	4.3E–08
Cadmium	SSS-009	23	2.3E–06	1.1E–01	2.5E–07
Chromium (total)	SSS-009	1,500	1.5E–04	1.1E–01	1.6E–05
Cobalt	ALPE	22	2.2E–06	1.1E–01	2.4E–07
Copper	SSS-009	530	5.2E–05	1.1E–01	5.7E–06
Lead	SSD-009	320	3.1E–05	1.1E–01	3.4E–06
Mercury	SSD-009	20	2.0E–06	1.1E–01	2.2E–07
Molybdenum	SSS-009	16	1.6E–06	1.1E–01	1.7E–07
Nickel	SSS-009	670	6.6E–05	1.1E–01	7.2E–06
Silver	SSS-009	7.4	7.3E–07	1.1E–01	8.0E–08
Zinc	SSD-009	750	7.4E–05	1.1E–01	8.1E–06

[a]The total threshold limit concentration (TTLC) is determined by nitric acid digestion (see DHS, 1984).

[b]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in Section 3, the locations of samples SSS-009 and SSD-009 have been remediated.

Appendix Table A-5. Total equivalent lifetime soil-dermal exposure of adults onsite (aos), $e^{org}_{3\text{-}max}$(aos), for a 50-y period for each of the 20 organic chemicals in soil at the LLNL site with a frequency of detection (>LOD) that is greater than or equal to 5%.

Organic chemical	Location[a]	[$C^{org}_{s\text{-}max}$] Maximum concentration in soil (mg/kg)	[F_3] PEF for dermal absorption of soil (mg/kg-d per mg/kg)	[$e^{org}_{3\text{-}max}$] Dermal exposure for adults onsite (mg/kg-d)
Acetone	SSS-009	8.0E+00	1.3E–06	1.0E–05
Aroclor (PCB) 1254	SSD-008	1.3E+00	1.3E–06	1.7E–06
Benzene	ALPW	1.1E–02	1.3E–06	1.4E–08
Chloroform	ASS	9.5E–03	1.3E–06	1.2E–08
1,2-dichlorobenzene	ASN	8.7E–03	1.3E–06	1.1E–08
1,2-dichloroethylene (total 1,2-DCE)	ALPE	1.6E–03	1.3E–06	2.1E–09
Ethylbenzene	ALPW	5.2E–03	1.3E–06	6.8E–09
Fluoranthene	SSS-009	1.8E+00	1.3E–06	2.3E–06
Methyl acetate	SSD-006	2.0E+00	1.3E–06	2.6E–06
Methylene chloride (dichloromethane)	161-2	5.0E–02	1.3E–06	6.5E–08
Methyl ethyl ketone	612-S2, 612-S3	4.0E–01	1.3E–06	5.2E–07
Phenanthrene	SSS-009	1.0E+00	1.3E–06	1.3E–06
Pyrene	SSS-009	1.3E+00	1.3E–06	1.7E–06
Tetrachloroethylene (PCE)	518-1	3.7E–01	1.3E–06	4.8E–07
Toluene	ALPE	8.3E–03	1.3E–06	1.1E–08
1,1,1-trichloroethane (1,1,1-TCA)	513-1	1.4E–01	1.3E–06	1.8E–07
Trichloroethylene (TCE)	518-2	3.0E+00	1.3E–06	3.9E–06
Trichlorofluoromethane (Freon 11)	SSD-013, SSD-014	3.0E–01	1.3E–06	3.9E–07
Trichlorotrifluoroethane (Freon 113)	513-1	1.6E–01	1.3E–06	2.1E–07
Xylene isomers (total)	SSD-006, SSD-010, SSD-013, SSD-014	2.0E–01	1.3E–06	2.6E–07

[a]See Figures 3-12 and 3-13 for sampling locations. As noted in Section 3, the locations of samples SSS-009 and SSD-008 have been remediated.

Appendix Table A-6. Total equivalent lifetime soil-dermal exposure of adults onsite (aos), $[e^{inorg}_{3\text{-}max}](aos)$, for a 50-y period for each of the inorganic chemicals in soil at the LLNL site with a maximum concentration (TTLC[a]) distinct from background levels (as discussed in Appendix C).

Chemical	Location[b]	$[C^{inorg}_{s\text{-}max}]$ Maximum soil concentration at LLNL (mg/kg)	$[F_3]$ PEF for dermal absorption of soil (mg/kg-d per mg/kg)	$[e^{inorg}_{3\text{-}max}]$ Dermal exposure for adults onsite (mg/kg-d)
Antimony	SSS-009	13	1.3E–06	1.7E–05
Arsenic	SSS-003	14	1.3E–06	1.8E–05
Beryllium	SSD-009	4	1.3E–06	5.2E–06
Cadmium	SSS-009	23	1.3E–06	3.0E–03
Chromium (total)	SSS-009	1,500	1.3E–06	2.0E–03
Cobalt	ALPE	22	1.3E–06	2.9E–05
Copper	SSS-009	530	1.3E–06	6.9E–04
Lead	SSD-009	320	1.3E–06	4.2E–04
Mercury	SSD-009	20	1.3E–06	2.6E–05
Molybdenum	SSS-009	16	1.3E–06	2.1E–05
Nickel	SSS-009	670	1.3E–06	8.7E–04
Silver	SSS-009	7.4	1.3E–06	9.6E–06
Zinc	SSD-009	750	1.3E–06	9.8E–04

[a]The TTLC is determined by nitric acid digestion (see DHS, 1984).

[b]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in Section 3, the locations of samples SSS-009 and SSD-009 have been remediated.

Appendix Table A-7. Soil-based pathway-exposure factors and corresponding environmental concentration parameters applicable to chemicals identified to be of concern in soil on the LLNL site.

Exposure route	Pathway exposure factor (PEF)	Concentration ($C_{i\text{-}max}$)	
		Parameter	Units
Ingestion	$F_{1\text{-}max}$(aos) = 5.4×10^{-7} mg/kg-d per mg/kg		mg/kg
Inhalation of soil particles	$F_{2a\text{-}max}$(aos) = 1.1×10^{-1} mg/kg-d per mg/m^3	$C_{p\text{-}max}$	mg/m^3
Inhalation of VOCs volatilized from soil	$F_{2b\text{-}max}$(aos) = 5.2×10^{-2} mg/kg-d per mg/m^3	$C_{a\text{-}max}$(aos)	mg/m^3
	$F_{2b\text{-}max}$(public) = 8.2×10^{-2} mg/kg-d per mg/m^3	$C_{a\text{-}max}$(public)	mg/m^3
Dermal absorption	F_3(aos) = 1.3×10^{-6} mg/kg-d per mg/kg	$C_{s\text{-}max}$	mg/kg

Appendix Table A-8. Total equivalent lifetime soil-based exposure of adults onsite E^{org}_{max}(aos), to concentrations of each of the 20 organic chemicals in soil at the LLNL site with a frequency of detection (>LOD) that is greater than or equal to 5%.

		Soil-based exposure for adults onsite			
Organic chemical	Location[a]	$[e^{org}_{1\text{-}max}]$ Ingestion (mg/kg-d)	$[e^{org}_{2a\text{-}max}]$ Particle inhalation (mg/kg-d)	$[e^{org}_{3\text{-}max}]$ Dermal absorption (mg/kg-d)	$[E^{org}_{s\text{-}max}]$ Total lifetime (mg/kg-d)
Acetone	SSS-009	4.3E–06	8.6E–08	1.0E–05	1.5E–05
Aroclor (PCB) 1254	SSD-008	7.0E–07	1.4E–08	1.7E–06	2.4E–06
Benzene	ALPW	5.9E-09	1.2E–10	1.4E–08	2.0E–08
Chloroform	ASS	5.1E–09	1.0E–10	1.2E–08	1.8E–08
1,2-dichlorobenzene	ASN	4.7E–09	9.4E–11	1.1E–08	1.6E–08
1,2-dichloroethylene (total 1,2-DCE)	ALPE	8.6E–10	1.7E–11	2.1E–09	3.0E–09
Ethylbenzene	ALPW	2.8E–09	5.6E–11	6.8E–09	9.6E–09
Fluoranthene	SSS-009	9.7E–07	1.9E–08	2.3E–06	3.3E–06
Methyl acetate	SSD-006	1.1E–06	2.2E–08	2.6E–06	3.7E–06
Methylene chloride (dichloromethane)	161-2	2.7E–08	5.4E–10	6.5E–08	9.3E–08
Methyl ethyl ketone	612-S2, 612-S3	2.2E–07	4.3E–09	5.2E–07	7.4E–07
Phenanthrene	SSS-009	5.4E–07	1.1E–08	1.3E–06	1.9E–06
Pyrene	SSS-009	7.0E–07	1.4E–08	1.7E–06	2.4E–06
Tetrachloroethylene (PCE)	518-1	2.0E–07	4.0E–09	4.8E–07	6.8E–07
Toluene	ALPE	4.5E–09	8.9E–11	1.1E–08	1.5E–08
1,1,1-trichloroethane (1,1,1-TCA)	513-1	7.6E–08	1.5E–09	1.8E–07	2.6E–07
Trichloroethylene (TCE)	518-2	1.6E–06	3.2E–08	3.9E–06	7.9E–06[b]
Trichlorofluoromethane (Freon 11)	SSD-013, SSD-014	1.6E–07	3.2E–09	3.9E–07	5.6E–07
Trichlorotrifluoroethane (Freon 113)	513-1	8.6E–08	1.7E–09	2.1E–07	3.0E–07
Xylene isomers (total)	SSD-006, SSD-010, SSD-013, SSD-014	1.1E–07	2.2E–09	2.6E–07	3.7E–07

[a]See Figures 3-12 and 3-13 for sampling locations. As noted in Section 3, the locations of samples SSS-009 and SSD-008 have been remediated.

[b]Includes value for $e^{org}_{2b\text{-}max}$(aos) equal to 2.4×10^{-6} mg/kg-d, which is based on a value for $C_{a\text{-}max}$(aos) equal to 4.6×10^{-5} mg/m^3 and a value for F_{2b}(aos) equal to 5.2×10^{-2} m^3/kg-d.

Appendix Table A-9. Total equivalent lifetime soil-based exposure to concentrations of inorganic chemicals in soil at the LLNL site distinct from background levels by adults onsite, $E^{inorg}_{s\text{-}max}$(aos), for a continuous 50-y period.

Inorganic chemical	$[e^{inorg}_{1\text{-}max}]$ Ingestion (mg/kg-d)	$[e^{inorg}_{2a\text{-}max}]$ Particle inhalation (mg/kg-d)	$[e^{inorg}_{3\text{-}max}]$ Dermal absorption (mg/kg-d)	$[E^{inorg}_{s\text{-}max}]$ Total lifetime (mg/kg-d)
Antimony	7.0E–06	1.4E–07	1.7E–05	2.4E–05
Arsenic	7.6E–06	1.5E–07	1.8E–05	2.6E–05
Beryllium	2.2E–06	4.3E–08	5.2E–06	7.4E–06
Cadmium	1.2E–05	2.5E–07	3.0E–05	4.3E–05
Chromium (total)	8.1E–04	1.6E–05	2.0E–03	2.8E–03
Cobalt	1.2E–05	2.4E–07	2.9E–05	4.1E–05
Copper	2.9E–04	5.7E–06	6.9E–04	9.8E–04
Lead	1.7E–04	3.4E–06	4.2E–04	5.9E–04
Mercury	1.1E–05	2.2E–07	2.6E–05	3.7E–05
Molybdenum	8.6E–06	1.7E–07	2.1E–05	3.0E–05
Nickel	3.6E–04	7.2E–06	8.7E–04	1.2E–03
Silver	4.0E–06	8.0E–08	9.6E–06	1.4E–05
Zinc	4.1E–04	8.1E–06	9.8E–04	1.4E–03

Appendix B

Comparison of EPA and LLNL Methods for Deriving Carcinogenic Risk and Noncarcinogenic Hazard Associated with Soil Constituents

Appendix B

Comparison of EPA and LLNL Methods for Deriving Carcinogenic Risk and Noncarcinogenic Hazard Associated with Soil Constituents

J. I. Daniels

The soil-based exposure and screening risk assessments presented in Section 3 (see also Appendix A) were obtained using a procedure similar to that recommended by the EPA. The results are somewhat different than those obtained using EPA methodology (U.S. EPA, 1986, 1989a). The principal differences between the two methods for estimating exposure and screening risk are the way exposure is derived for each exposure pathway and the manner in which an applicable cancer-potency factor (CPF) or reference dose (RfD) for the respective calculations of carcinogenic risk and noncarcinogenic hazard is applied. For example, the EPA method for calculating maximum exposures is based on default parameters that typically result in dermal uptake being virtually equal to oral intake. This is not the case in the approach used by the LLNL, which is based on more realistic assumptions with regard to parameters for estimating exposure by dermal contact and by oral intake. Moreover, the LLNL method uses maxima for CPFs and RfDs to achieve conservative estimates of maximum values of carcinogenic risk and noncarcinogenic hazard for screening. Alternatively, the EPA uses route-of-exposure-specific CPFs and RfDs for screening.

In addition to the above differences between EPA and LLNL methods, the LLNL procedure for identifying the inorganic chemicals of concern also differs from that typically used by the EPA. In the LLNL procedure, background levels of inorganic chemicals are distinguished from concentrations representing contamination from a local source by interpreting the statistical distribution of the concentration data. The data are plotted graphically and analyzed according to the procedure described by Michels (1971), which is explained in Appendix C. Alternatively, EPA methodology allows comparisons to be made between maximum detected concentrations and reported ranges of local or regional concentrations in order to make the same distinction between contamination and background levels. Accordingly, in this appendix, those inorganic chemicals identified by the LLNL method as being of concern in onsite soil are addressed using EPA methodology. These results are then compared with those obtained for the same inorganic chemicals using the LLNL procedure.

Appendix Tables B-1 and B-2 address the noncarcinogenic hazard and carcinogenic risk, respectively, for the organic chemicals of concern that were calculated using the U.S. EPA procedure. For comparison, these tables also contain the respective values computed with the LLNL procedure (Table 3-8 of Section 3). Appendix Tables B-3 through B-5 present similar calculations and comparisons for the EPA procedure. Although the EPA methodology tends to yield higher values for screening risk and hazard, we feel that results obtained from the two

methods are in reasonable agreement. In the one case where there is a significant difference (Appendix Table B-5), the same chemical (chromium) dominates the screening risk.

Appendix B References

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Appendix Table B-1. Screening analyses based on EPA methodology for calculating for a continuous 50-y period. Total hazard is compared to similar value calculated by

Inorganic chemicals at concentrations distinct from background levels	Maximum soil concentration (mg/kg)	Location[a]	Maximum oral intake, MOI (mg/kg-d)[b]	Reference dose for oral intake, RfD_o (mg/kg-d)[c]	Hazard index for maximum oral intake, HI_o (MOI/RfD_o)
Acetone	8.0E+00	SSS-009	2.3E–05	1.0E–01	2.3E–04
Aroclor (PCB) 1254	1.3E+00	SSD-008	3.7E–06	1.0E–03[f]	3.7E–03
Benzene	1.1E–02	ALPW	3.1E–08	(h)	—
Chloroform	9.5E–03	ASS	2.7E–08	1.0E–02	2.7E–06
1,2-dichlorobenzene	8.7E–03	ASN	2.5E–08	8.9E–02	2.8E–07
1,2-dichloroethylene (total)	1.6E–03	ALPE	4.6E–09	2.0E–02[g]	2.3E–07
Ethylbenzene	5.2E–03	ALPW	1.5E–08	1.0E–01	1.5E–07
Fluoranthene	1.8E+00	SSS-009	5.1E–06	(h)	
Methyl acetate	2.0E+00	SSD-006	5.7E–06	1.9E+01	3.0E–07
Methylene chloride (dichloromethane)	5.0E–02	161–2	1.4E–07	6.0E–02	2.4E–06
Methyl ethyl ketone	4.0E–01	612–S2, 612–S3	1.1E–06	5.0E–02	2.3E–05
Phenanthrene	1.0E+00	SSS-009	2.9E–06	(h)	—
Pyrene	1.3E+00	SSS-009	3.7E–06	(h)	—
Tetrachloroethylene (PCE)	3.7E–01	518–1	1.1E–06	1.0E–02	1.1E–04
Toluene	8.3E–03	ALPE	2.4E–08	3.0E–01	7.9E–08
1,1,1-trichloroethane (1,1,1-TCA)	1.4E–01	513–1	4.0E–07	9.0E–02	4.4E–06
Trichloroethylene (TCE)	3.0E+00	518–2	8.6E–06	(h)	—
Trichlorofluoromethane (Freon 11)	3.0E–01	SSD-013, SSD-014	8.6E–07	3.0E–01	2.9E–06
Trichlorotrifluorethane (Freon 113)	1.6E–01	513–1	4.6E–07	3.0E+01	1.5E–08
Xylene isomers (total)	2.0E–01	SSD-006, SSD-010, SSD-013, SSD-014	5.7E–07	9.0E–02	6.3E–06
Hazard index totals					4.1E–03

potential hazard resulting from soil-based exposures to organic chemicals by adults onsite LLNL procedure (see Table 3-8).

Maximum dermal uptake, MDU (mg/kg-d)[d]	Reference dose for dermal uptake, RfD_d (mg/kg-d)[c]	Hazard index for dermal uptake, HI_d (MDU/RfDd)	Maximum inhalation intake, MII (mg/kg-d)[e]	Reference dose for inhalation intake, RfD_h (mg/kg-d)[c]	Hazard index for maximum inhalation intake, HI_h (MII/RfDh)	Total hazard index determined by EPA method ($HI_o + HI_d + HI_h$)	Total hazard index determined by LLNL method
2.3E–05	1.0E–01	2.3E–04	1.6E–07	1.0E–01	1.6E–06	4.6E–04	1.5E–04
3.7E–06	1.0E–03	3.7E–03	2.6E–08	1.0E–03[f]	2.6E–05	7.5E–03	2.4E–03
3.1E–08	[h]	—	2.2E–10	[h]	—	—	—
2.7E–08	1.0E–02	2.7E–06	1.9E–10	1.0E–02	1.9E–08	5.4E–06	1.8E–06
2.5E–08	8.9E–02	2.8E–07	1.7E–10	8.9E–02	2.0E–09	5.6E–07	1.8E–07
4.6E–09	2.0E–02	2.3E–07	3.2E–11	2.0E–02[g]	1.6E–09	4.6E–07	1.5E–07
1.5E–08	1.0E–01	1.5E–07	1.0E–10	1.0E–01	1.0E–09	3.0E–07	8.7E–08
5.1E–06	[h]	—	3.6E–08	[h]	—	—	—
5.7E–06	1.9E+01	3.0E–07	4.0E–08	1.9E+01	2.1E–09	6.0E–07	1.9E–07
1.4E–07	6.0E–02	2.4E–06	1.0E–09	9.0E–01	1.1E–09	4.8E–06	1.0E–07
1.1E–06	5.0E–02	2.3E–05	8.0E–09	9.0E–02	8.9E–08	4.6E–05	8.2E–06
2.9E–06	[h]	—	2.0E–08	[h]	—	—	—
3.7E–06	[h]	—	2.6E–08	[h]	—	—	—
1.1E–06	1.0E–02	1.1E–04	7.4E–09	1.0E–02	7.4E–07	2.1E–04	6.8E–05
2.4E–08	3.0E–01	7.9E–08	1.7E–10	6.0E–01	2.8E–10	1.6E–07	2.5E–08
4.0E–07	9.0E–02	4.4E–06	2.8E–09	3.0E–01	9.3E–09	8.9E–06	8.7E–07
8.6E–06	[h]	—	6.0E–08	[h]	—	—	—
8.6E–07	3.0E–01	2.9E–06	6.0E–09	2.0E–01	3.0E–08	5.7E–06	1.9E–06
4.6E–07	3.0E+01	1.5E–08	3.2E–09	3.0E+01	1.1E–10	3.1E–08	1.0E–08
5.7E–07	9.0E–02	6.3E–06	4.0E–09	2.0E+00	2.0E–09	1.3E–05	1.9E–07
		4.1E–03			2.8E–05	8.2E–03	2.6E–03

Appendix Table B-1. (Continued)

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in Section 3, the locations of samples SSS-009 and SSD-008 have been remediated.

[b]MOI = maximum soil concentration (i.e., mg/kg) × maximum soil ingestion rate for an adult (200 mg/d) × 10^{-6} kg of soil per mg of soil × GI-tract absorption (i.e., 1.0) × 1/70 kg of body weight.

[c]Route-specific RfD from available data in U.S. EPA 1989b and/or 1990 unless otherwise noted; RfDs for dermal uptake—and in most cases (unless otherwise stated), for inhalation intake—correspond to those for oral intake.

[d]MDU = maximum soil concentration (i.e., mg/kg) × maximum exposed skin area for an adult (1980 cm^2) × 10^{-6} kg of soil per mg of soil × maximum soil contact rate (i.e., 0.5) × dermal absorption (i.e., 0.2) × 1/70 kg of body weight.

[e]MII = maximum soil concentration (i.e., mg/kg) × concentration of airborne soil particles (i.e., 0.07 mg of soil per m^3 of air) × maximum soil inhalation rate for an adult (20 m^3/d) × 10^{-6} kg of soil per mg of soil × inhalation absorption (i.e., 1.0) × 1/70 kg of body weight.

[f]The RfD is based on an acceptable daily intake (ADI) for a newborn cited by Geyer *et al.* (1986).

[g]For trans-1,2-DCE isomer and applied to total 1,2-DCE.

[h]RfD is pending or no data available.

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The following table has facing pages.

Appendix Table B-2. Screening analyses based on EPA methodology for calculating adults onsite for a continuous 50-y period. Total carcinogenic risk is compared to similar

Inorganic chemicals at concentrations distinct from background levels	Maximum soil concentration (mg/kg)	Location[a]	Maximum oral intake, MOI (mg/kg-d)[b]	Cancer-potency factor for oral intake, CPF_o (mg/kg-d)[c]	Excess cancer risk for maximum oral intake, R_o ($MOI \times CPF_o$)
Acetone	8.0E+00	SSS-009	2.3E–05	(g)	—
Aroclor (PCB) 1254	1.3E+00	SSD-008	3.7E–06	7.7E+00	2.9E–05
Benzene	1.1E–02	ALPW	3.1E–08	2.9E–02	9.1E–10
Chloroform	9.5E–03	ASS	2.7E–08	6.1E–03	1.7E–10
1,2-dichlorobenzene	8.7E–03	ASN	2.5E–08	(g)	—
1,2-dichloroethylene (total)	1.6E–03	ALPE	4.6E–09	(g)	—
Ethylbenzene	5.2E–03	ALPW	1.5E–08	Group D	—
Fluoranthene	1.8E+00	SSS-009	5.1E–06	(g)	—
Methyl acetate	2.0E+00	SSD-006	5.7E–06	(g)	—
Methylene chloride (dichloromethane)	5.0E–02	161–2	1.4E–07	7.5E–03	1.1E–09
Methyl ethyl ketone	4.0E–01	612–S2, 612–S3	1.1E–06	Group D	—
Phenanthrene	1.0E+00	SSS-009	2.9E–06	(g)	—
Pyrene	1.3E+00	SSS-009	3.7E–06	(g)	—
Tetrachloroethylene (PCE)	3.7E–01	518–1	1.1E–06	5.1E–02	5.4E–08
Toluene	8.3E–03	ALPE	2.4E–08	Group D	—
1,1,1-trichloroethane (1,1,1-TCA)	1.4E–01	513–1	4.0E–07	Group D	—
Trichloroethylene (TCE)	3.0E+00	518–2	8.6E–06	1.1E–02	9.4E–08
Trichlorofluoromethane (Freon 11)	3.0E–01	SSD-013, SSD-014	8.6E–07	(g)	—
Trichlorotrifluorethane (Freon 113)	1.6E–01	513–1	4.6E–07	(g)	—
Xylene isomers (total)	2.0E–01	SSD-006, SSD-010, SSD-013, SSD-014	5.7E–07	Group D	—
Screening risk totals					2.9E–05

potential carcinogenic risk resulting from soil-based exposures to organic chemicals by value calculated by LLNL procedure (see Table 3-8).

Maximum dermal uptake, MDU (mg/kg-d)[d]	Cancer-potency factor for dermal uptake, CPFd (mg/kg-d)[e]	Excess cancer risk for maximum dermal uptake, Rd (MDU × CPFd)	Maximum inhalation intake, MII (mg/kg-d)[f]	Cancer-potency factor for inhalation intake, CPFh (mg/kg-d)[c]	Excess cancer risk for maximum inhalation intake, Rh (MII × CPFd)	Total excess cancer risk by EPA method (Ro + Rd + Rh)	Total excess cancer risk by LLNL method
2.3E–05	(g)	—	1.6E–07	(g)	—	—	—
3.7E–06	7.7E+00	2.8E–05	2.6E–08	7.7E+00	2.0E–07	5.7E–05[h]	1.8E–05[h]
3.1E–08	2.9E–02	9.0E–10	2.2E–10	2.9E–02	6.4E–12	1.8E–09	5.8E–10
2.7E–08	6.1E–03	1.6E–10	1.9E–10	8.1E–02	1.5E–11	3.4E–10	1.5E–09
2.5E–08	(g)	—	1.7E–10	(g)	—	—	—
4.5E–09	(g)	—	3.2E–11	(g)	—	—	—
1.5E–08	Group D	—	1.0E–10	Group D	—	—	—
5.1E–06	(g)	—	3.6E–08	(g)	—	—	—
5.7E–06		—	4.0E–08	(g)	—	—	—
1.4E–07	7.5E–03	1.1E–09	1.0E–09	1.7E–03	1.7E–12	2.1E–09	7.0E–10
1.1E–06	Group D	—	8.0E–09	Group D	—	—	—
2.8E–06	(g)	—	2.0E–08	(g)	—	—	—
3.7E–06	(g)	—	2.6E–08	(g)	—	—	—
1.0E–06	5.1E–02	5.3E–08	7.4E–09	3.3E–03	2.4E–11	1.1E–07	3.5E–08
2.3E–08	Group D	—	1.7E–10	Group D	—	—	—
4.0E–07	Group D	—	2.8E–09	Group D	—	—	—
8.5E–06	1.1E–02	9.3E–08	6.0E–08	1.7E–02	1.0E–09	1.9E–07	1.3E–07
8.5E–07	(g)	—	6.0E–09	(g)	—		
4.5E–07	(g)	—	3.2E–09	(g)	—	—	—
5.7E–07	Group D	—	4.0E–09	Group D	—	—	—
		2.8E–05			2.0E–07	5.7E–05	1.9E–05

Appendix Table B-2. (Continued)

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in Section 3, the locations for samples SSS-009 and SSD-008 have been remediated.

[b]MOI = maximum soil concentration (i.e., mg/kg) × maximum soil ingestion rate for an adult (200 mg/d) × 10^{-6} kg of soil per mg of soil × GI-tract absorption (i.e., 1.0) × 1/70 kg of body weight.

[c]Route-specific CPF from available data in U.S. EPA 1989b and/or 1990.

[d]MDU = maximum soil concentration (i.e., mg/kg) × maximum exposed skin area for an adult (1980 cm^2) × 10^{-6} kg of soil per mg of soil × maximum soil contact rate (i.e., 0.5) × dermal absorption (i.e., 0.2) × 1/70 kg of body weight.

[e]CPF for dermal-exposure pathway corresponds to CPF for oral intake.

[f]MII = maximum soil concentration (i.e., mg/kg) × concentration of airborne soil particles (i.e., 0.07 mg of soil per m^3 of air) × maximum soil inhalation rate for an adult (20 m^3/d) × 10^{-6} kg of wt/mg of soil × inhalation absorption (i.e., 1.0) × 1/70 kg of body weight.

[g]CPF is pending, under review, or no data available.

[h]All contaminated material at location SSD-008 has been removed and properly disposed of. The location with the highest *existing* concentration of Aroclor 1254 is SSD-001, with 3.5E–01 mg/kg. For this concentration, the EPA screening value is 1.5E–05 and the LLNL value is 4.8E–06.

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The following table has facing pages.

Appendix Table B-3. Screening analyses based on EPA methodology for calculating chemicals by adults onsite for a continuous 50-y period. Total noncarcinogenic hazard is

Inorganic chemicals at concentrations distinct from background levels	Maximum soil concentration (mg/kg)	Location[a]	Maximum oral intake, MOI (mg/kg-d)[b]	Reference dose for oral intake, RfD_o (mg/kg-d)[c]	Hazard index for maximum oral intake, HI_o (MOI/RfD_o)
Antimony	13	SSS-009	3.7E–05	4.0E–04	9.3E–02
Arsenic	14	SSS-003	4.0E–05	1.0E–03	4.0E–02
Beryllium	4	SSD-009	1.1E–05	5.0E–03	2.3E–03
Cadmium	23	SSS-009	6.6E–05	5.0E–04	1.3E–01
Chromium (total)	1500[j]	SSS-009	4.3E–03	5.0E–03[f]	8.6E–01
Cobalt	22	ALPE	6.3E–05	1.6E+00[g]	3.9E–05
Copper	530	SSS-009	1.5E–03	2.9E–02[h]	5.2E–02
Lead	320[k]	SSD-009	9.1E–04	1.4E–03[h]	6.5E–01
Mercury	20[l]	SSD-009	5.7E–05	3.0E–04	1.9E–01
Molybdenum	16	SSS-009	4.6E–05	2.1E–03[i]	2.2E–02
Nickel	670	SSS-009	1.9E–03	2.0E–02	9.6E–02
Silver	7.4	SSS-009	2.1E–05	3.0E–03	7.0E–03
Zinc	750	SSS-009	2.1E–03	2.0E–01	1.1E–02
Hazard index totals					2.2E+00

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found.

[b]MOI = maximum soil concentration (i.e., mg/kg) × maximum soil ingestion rate for an adult (200 mg/d) × 10^{-6} kg of soil per mg of soil × GI-tract absorption (i.e., 1.0) × 1/70 kg of body weight.

[c]Route-specific RfD from available data in U.S. EPA 1989b and/or 1990 unless otherwise noted. These RfDs are used in calculations involving dermal uptake and inhalation intake.

[d]MDU = maximum soil concentration (i.e., mg/kg) × maximum exposed skin area for an adult (1980 cm^2) × 10^{-6} kg of soil per mg of soil × maximum soil contact rate (i.e., 0.5) × dermal absorption (i.e., 0.2) × 1/70 kg of body weight.

[e]MII = maximum soil concentration (i.e., mg/kg) × concentration of airborne soil particles (i.e., 0.07 mg of soil per m^3 of air) × maximum soil inhalation rate for an adult (20 m^3/d) × 10^{-6} kg of soil per mg of soil × inhalation absorption (i.e., 1.0) × 1/70 kg of body weight.

[f]Oral RfD for chromium (VI) is used (see IRIS data base; U.S. EPA, 1990).

[g]Estimated acceptable daily intake (ADI) based on evidence reported in the literature that $CoSO_4$ has been used therapeutically at up to 300 mg/d without any symptoms of adverse health effects (National Research Council Safe Drinking Water Committee, 1977). Consequently, the ADI corresponds to a daily dose of cobalt of 114 mg/d divided by 70 kg.

potential noncarcinogenic hazard resulting from soil-based exposures to inorganic compared to similar value calculated by LLNL procedure (see Table 3-10).

Maximum dermal uptake, MDU (mg/kg–d)[d]	Reference dose for dermal uptake, RfD_d (mg/kg-d)[c]	Hazard index dermal uptake, HI_d (MDU/RfDd)	Maximum inhalation intake, MII (mg/kg-d)[e]	Reference dose for inhalation intake, RfD_h (mg/kg-d)[c]	Hazard index for maximum inhalation intake, HI_h (MII/RfDh)	Total hazard index determined by EPA method ($HI_o + HI_d + HI_h$)	Total hazard index determined by LLNL method
3.7E–05	4.0E–04	9.2E–02	2.6E–07	4.0E–04	6.5E–04	1.9E–01	6.0E–02
4.0E–05	1.0E–03	4.0E–02	2.8E–07	1.0E–03	2.8E–04	8.0E–02	2.6E–02
1.1E–05	5.0E–03	2.3E–03	8.0E–08	5.0E–03	1.6E–05	4.6E–03	1.5E–03
6.5E–05	5.0E–04	1.3E–01	4.6E–07	5.0E–04	9.2E–04	2.6E–01	8.6E–02
4.2E–03	5.0E–03[f]	8.5E–01	3.0E–05	5.0E–03[f]	6.0E–03	1.7E+00[j]	5.6E–01[j]
6.2E–05	1.6E+00[g]	3.9E–05	4.4E–07	1.6E+00[g]	2.8E–07	7.8E–05	2.6E–05
1.5E–03	2.9E–02[h]	5.2E–02	1.1E–05	2.9E–02[h]	3.7E–04	1.0E–01	3.4E–02
9.1E–04	1.4E–03[h]	6.5E–01	6.4E–06	1.4E–03[h]	4.6E–03	1.3E+00[k]	4.2E–01[k]
5.7E–05	3.0E–04	1.9E–01	4.0E–07	3.0E–04	1.3E–03	3.8E–01[l]	1.2E–01[l]
4.5E–05	2.1E–03[i]	2.2E–02	3.2E–07	2.1E–03[i]	1.5E–04	4.3E–02	1.4E–02
1.9E–03	2.0E–02	9.5E–02	1.3E–05	2.0E–02	6.7E–04	1.9E–01	6.0E–02
2.1E–05	3.0E–03	7.0E–03	1.5E–07	3.0E–03	4.9E–05	1.4E–02	4.7E–03
2.1E–03	2.0E–01	1.1E–02	1.5E–05	2.0E–01	7.5E–05	2.1E–02	7.0E–03
		2.1E+00			1.5E–02	4.3E+00	1.4E+00

[h]Estimated ADI based on Federal and State of California maximum contaminant level (MCL) for this chemical in drinking water (U.S. EPA, 1988a, and DHS 1989a, for lead; U.S. EPA, 1988b, and DHS, 1989b, for copper) and assuming a consumption rate of 2 L/d. Consequently, the ADI corresponds to the MCL multiplied by 2 L/d and divided by 70 kg.

[i]Estimated ADI based on the National Research Council Committee on Dietary Allowances (1980) establishing 0.15 to 0.5 mg/d as an estimate of the safe and adequate intake range for molybdenum. To be conservative, the lower value was used, and the ADI corresponds to the value divided by 70 kg.

[j]All contaminated materials at location SSS-009 have been removed and properly disposed of. The location with the highest *existing* concentration of total chromium is SSS-010, with 110 mg/kg. The associated EPA hazard index is 1×10^{-1}, and the LLNL hazard index is 4.1E–02.

[k]All contaminated materials at location SSD-009 have been removed and properly disposed of. The location with the highest *existing* concentration of lead is SSD-002, with 130 mg/kg. The associated EPA hazard index is 5.2×10^{-1}, and the LLNL hazard index is 1.7E–01.

[l]All contaminated materials at location SSD-009 have been removed and properly disposed of. The location with the highest *existing* concentration of mercury is SSD-002, with 0.4 mg/kg. The associated EPA hazard index is 7.6×10^{-3}, and the LLNL hazard index is 2.4E–02.

Appendix Table B-4. Screening analyses based on EPA methodology for calculating chemicals by adults onsite for a continuous 50-y period, arranged by target organ. LLNL procedure (see Table 3-11).

Inorganic chemicals at concentrations distinct from background levels	Maximum soil concentration (mg/kg)	Location[a]	Maximum oral intake, MOI (mg/kg-d)[b]	Reference dose for oral intake, RfD_o (mg/kg-d)[c]	Hazard index for maximum oral intake, HI_o (MOI/RfD_o)
Antimony (target organ is blood)	13	SSS-009	3.7E–05	4.0E–04	9.3E–02
Zinc (target organ is blood)	750	SSS-009	2.1E–03	2.0E–01	1.1E–02
Hazard index subtotal					1.0E–01
Arsenic (skin is target)	14	SSS-003	4.0E–05	1.0E–03	4.0E–02
Beryllium (target not determined)	4	SSD-009	1.1E–05	5.0E–03	2.3E–03
Cadmium (kidney is target)	23	SSS-009	6.6E–05	5.0E–04	1.3E–01
Chromium (total) (reduced water consumption)	1500	SSS-009	4.3E–03	5.0E–03[f]	8.6E–01
Cobalt (target not determined)	22	ALPE	6.3E–05	1.6E+00[g]	3.9E–05
Copper (gastrointestinal tract is target)	530	SSS-009	1.5E–03	2.9E–02[h]	5.2E–02
Molybdenum (target not determined)	16	SSS-009	4.6E–05	2.1E–03[i]	2.2E–02
Nickel (reduced organ and body weights)	670	SSS-009	1.9E–03	2.0E–02	9.6E–02
Hazard index subtotal					1.2E+00
Lead (central nervous system is target)	320	SSD-009	9.1E–04	1.4E–03[h]	6.5E–01
Mercury (central nervous system is target)	20	SSD-009	5.7E–05	3.0E–04	1.9E–01
Silver (brain)	7.4	SSS-009	2.1E–05	3.0E–03	7.0E–03
Hazard index subtotal					2.0E–01
Hazard index grand total					1.5E+00

potential noncarcinogenic hazard resulting from soil-based exposures to inorganic subtotals and total noncarcinogenic hazard are compared to similar values calculated by

Maximum dermal uptake, MDU (mg/kg-d)[d]	Reference dose for dermal uptake, RfD_d (mg/kg-d)[c]	Hazard index dermal uptake, HI_d (MDU/RfDd)	Maximum inhalation intake, MII (mg/kg-d)[e]	Reference dose for inhalation intake, RfD_h (mg/kg-d)[c]	Hazard index for maximum inhalation intake, HI_h (MII/RfDh)	Total hazard index determined by EPA method ($HI_o + HI_d + HI_h$)	Total hazard index determined by LLNL method
3.7E–05	4.0E–04	9.2E–02	2.6E–07	4.0E–04	6.5E–04	1.9E–01	6.0E–02
2.1E–03	2.0E–01	1.1E–02	1.5E–05	2.0E–01	7.5E–05	2.1E–02	7.0E–03
		1.0E–01			7.3E–04	2.1E–01	6.7E–02
4.0E–05	1.0E–03	4.0E–02	2.8E–07	1.0E–03	2.8E–04	8.0E–02	2.6E–02
1.1E–05	5.0E–03	2.3E–03	8.0E–08	5.0E–03	1.6E–05	4.6E–03	1.5E–03
6.5E–05	5.0E–04	1.3E–01	4.6E–07	5.0E–04	9.2E–04	2.6E–01	8.6E–02
4.2E–03	5.0E–03[f]	8.5E–01	3.0E–05	5.0E–03[f]	6.0E–03	1.7E+00	5.6E–01
6.2E–05	1.6E+00[g]	3.9E–05	4.4E–07	1.6E+00[g]	2.8E–07	7.8E–05	2.6E–05
1.5E–03	2.9E–02[h]	5.2E–02	1.1E–05	2.9E–02[h]	3.7E–04	1.0E–01	3.4E–02
4.5E–05	2.1E–03[i]	2.2E–02	3.2E–07	2.1E–03[i]	1.5E–04	4.3E–02	1.4E–02
1.9E–03	2.0E–02	9.5E–02	1.3E–05	2.0E–02	6.7E–04	1.9E–01	6.0E–02
		1.2E+00			8.4E–03	2.4E+00	7.8E–01
9.1E–04	1.4E–03[h]	6.5E–01	6.4E–06	1.4E–03[h]	4.6E–03	1.3E+00	4.2E–01
5.7E–05	3.0E–04	1.9E–01	4.0E–07	3.0E–04	1.3E–03	3.8E–01	1.2E–01
2.1E–05	3.0E–03	7.0E–03	1.5E–07	3.0E–03	4.9E–05	1.4E–02	4.7E–03
		2.0E–01			6.0E–03	1.7E+00	5.4E–01
		1.5E+00			1.5E–02	4.3E+00	1.4E+00

Appendix Table B-4. (Continued)

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found. As noted in Section 3, the locations of samples SSS-009 and SSD-009 have been remediated.

[b]MOI = maximum soil concentration (i.e., mg/kg) × maximum soil ingestion rate for an adult (200 mg/d) × 10^{-6} kg of soil per mg of soil × GI-tract absorption (i.e., 1.0) × 1/70 kg of body weight.

[c]Only oral reference dose (RfD) data are available from U.S. EPA, 1989b, and/or 1990, unless otherwise noted. These RfDs are used in calculations involving dermal uptake and inhalation intake.

[d]MDU = maximum soil concentration (i.e., mg/kg) × maximum exposed skin area for an adult (1980 cm^2) × 10^{-6} kg of soil per mg of soil × maximum soil contact rate (i.e., 0.5) × dermal absorption (i.e., 0.2) × 1/70 kg of body weight.

[e]MII = maximum soil concentration (i.e., mg/kg) × concentration of airborne soil particles (i.e., 0.07 mg of soil per m^3 of air) × maximum soil inhalation rate for an adult (20 m^3/day) × 10^{-6} kg of soil per mg of soil × inhalation absorption (i.e., 1.0) × 1/70 kg of body weight.

[f]Oral RfD for chromium (VI) is used (see IRIS data base; U.S. EPA, 1990).

[g]Estimated acceptable daily intake (ADI) based on evidence reported in the literature that $CoSO_4$ has been used therapeutically at up to 300 mg/d without any symptoms of adverse health effects (National Research Council Safe Drinking Water Committee, 1977). Consequently, the ADI corresponds to a daily dose of cobalt of 114 mg/d divided by 70 kg.

[h]Estimated ADI based on Federal and State of California MCLs for this chemical in drinking water (U.S. EPA, 1988a, and DHS 1989a, for lead; U.S. EPA, 1988b, and DHS, 1989b, for copper) and assuming a consumption rate of 2 L/d. Consequently, the ADI corresponds to the MCL multiplied by 2 L/d and divided by 70 kg.

[i]Estimated ADI based on the National Research Council Committee on Dietary Allowances (1980) establishing 0.15 to 0.5 mg/d as an estimate of the safe and adequate intake range for molybdenum. To be conservative, the lower value was used, and the ADI corresponds to the value divided by 70 kg.

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The following table has facing pages.

Appendix Table B-5. Screening analyses based on EPA methodology for calculating adults onsite for a continuous 50-y period. Total carcinogenic risk is compared to similar

Inorganic chemicals at concentrations distinct from background levels	Maximum soil concentration (mg/kg)	Location[a]	Maximum oral intake, MOI (mg/kg-d)[b]	Cancer-potency factor for oral intake, CPF_o (mg/kg-d)[c]	Excess cancer risk for maximum oral intake, R_o ($MOI \times CPF_o$)	Maximum dermal uptake, MDU (mg/kg-d)[d]
Antimony	13	SSS-009	3.7E–05	[h]	—	3.7E–05
Arsenic	14	SSS-003	4.0E–05	1.8E+00	7.2E–05	4.0E–05
Beryllium	4	SSD-009	1.1E–05	4.3E+00	4.9E–05	1.1E–05
Cadmium	23	SSS-009	6.6E–05	—	—	6.5E–05
Chromium (total)	1500[i]	SSS-009	4.3E–03	—	—	4.2E–03
Cobalt	22	ALPE	6.3E–05	[h]	—	6.2E–05
Copper	530	SSS-009	1.5E–03	[h]	—	1.5E–03
Lead	320	SSD-009	9.1E–04	[h]	—	9.1E–04
Mercury	20	SSD-009	5.7E–05	[h]	—	5.7E–05
Molybdenum	16	SSS-009	4.6E–05	[h]	—	4.5E–05
Nickel	670	SSS-009	1.9E–03	—	—	1.9E–03
Silver	7.4	SSS-009	2.1E–05	[h]	—	2.1E–05
Zinc	750	SSS-009	2.1E–03	[h]	—	2.1E–03
Risk totals					1.2E–04	

[a]Locations correspond to those shown in Figures 3-12 and 3-13, and are the sites where maximum concentrations were found.

[b]MOI = maximum soil concentration (i.e., mg/kg) × maximum soil ingestion rate for an adult (200 mg/d) × 10^{-6} kg of soil per mg of soil × GI-tract absorption (i.e., 1.0) × 1/70 kg of body weight.

[c]Route-specific cancer-potency factor specified or derived from unit-risk data in U.S EPA 1989b and/or 1990 unless otherwise noted.

[d]MDU = maximum soil concentration (i.e., mg/kg) × maximum exposed skin area for an adult (1980 cm^2) × 10^{-6} kg of soil per mg of soil × maximum soil contact rate (i.e., 0.5) × dermal absorption (i.e., 0.2) × 1/70 kg of body weight.

[e]CPF for dermal-exposure pathway corresponds to CPF for oral intake.

[f]MII = maximum soil concentration (i.e., mg/kg) × concentration of airborne soil particles (i.e., 0.07 mg of soil per m^3 of air) × maximum soil inhalation rate for an adult (20 m^3/d) × 10^{-6} kg of soil per mg of soil × inhalation absorption (i.e., 1.0) × 1/70 kg of body weight.

[g]Only total chromium was measured in soil samples but CPF is for chromium (VI), and chromium (VI) is presumed to be only a fraction of the total.

[h]Group D, noncarcinogenic, no data, or CPF not determined.

[i]All contaminated materials at locations SSS-009 and SSD-009 have been removed and properly disposed of. The location with the highest *existing* total chromium is SSS-010, with 110 mg/kg. The associated screening values for this concentration are 9.5×10^{-5} and 7.3×10^{-3} using the EPA and LLNL methodologies, respectively. The total screening risks for the EPA and LLNL methodologies are 4×10^{-4} and 9×10^{-3}, respectively.

potential carcinogenic risk resulting from soil-based exposures to inorganic chemicals by value calculated by LLNL procedure (see Table 3-12).

Cancer-potency factor for dermal uptake, CPF_d (mg/kg-d)[e]	Excess cancer risk for maximum dermal uptake, R_d (MDU × CPF_d)	Maximum inhalation intake, MII (mg/kg-d)[f]	Cancer-potency factor for inhalation intake, CPF_h (mg/kg-d)[c]	Excess cancer risk for maximum inhalation intake, R_h (MII × CPF_d)	Total excess cancer risk by EPA method ($R_o + R_d + R_h$)	Total excess cancer risk by LLNL method
[h]	—	2.6E–07	[h]	—	—	—
1.8E+00	7.2E–05	2.8E–07	1.5E+01	4.2E–06	1.5E–04	3.9E–04
4.3E+00	4.9E–05	8.0E–08	8.4E+00	6.7E–07	9.8E–05	6.2E–05
—	—	4.6E–07	6.3E+00	2.9E–06	2.9E–06	2.7E–04
—	—	3.0E–05	4.2E+01[g]	1.3E–03	1.3E–03[i]	1.2E–01[i]
[h]	—	4.4E–07	[h]	—	—	—
[h]	—	1.1E–05	[h]	—	—	—
[h]	—	6.4E–06	[h]	—	—	—
[h]	—	4.0E–07	[h]	—	—	—
[h]	—	3.2E–07	[h]	—	—	—
—	—	1.3E–05	8.4E–01	1.1E–05	1.1E–05	1.0E–03
[h]	—	1.5E–07	[h]	—	—	—
[h]	—	1.5E–05	[h]	—	—	—
	1.2E–04			1.3E–03	1.6E–03[i]	1.2E–01[i]

Appendix C

Comparisons of Maximum Soil Concentrations of Inorganic Chemicals on the LLNL Site with Those Reported for Selected Counties in California and Site 300

Appendix C

Comparisons of Maximum Soil Concentrations of Inorganic Chemicals on the LLNL Site with Those Reported for Selected Counties in California and Site 300

J. I. Daniels

Appendix Table C-1 in this appendix contains comparisons of maximum total threshold limit concentrations (TTLC) data for inorganic chemicals monitored in soil on the LLNL site and in nearby arroyos (see Figs. 3-12 and 3-13) with maximum TTLC values reported for soils at Site 300 and TTLC values reported for selected counties in California. Appendix Table C-2 contains the near-surface soil concentrations reported for Site 300. Site 300 soil-monitoring data is considered because of proximity to LLNL and absence of regional data for the immediate area surrounding LLNL.

This appendix also contains 23 graphs that are plots of the concentrations of inorganic substances (Figs. C-1 through C-17) and radioactive chemicals (Figs. C-18 through C-23) detected above the LOD on a linear y-axis scale (in units of the data) against a probability x-axis scale that is linear in units of standard deviation rather than in units of cumulative percent. These graphs were constructed in accordance with the procedure described by Michels (1971) for making distinctions between background levels of chemicals in soil samples and those that can be attributed to contamination. In this procedure all meaning comes from the relationships among values and so distributions are the primary objects that are described. For example, data are arranged in rank order and a percentile is computed for each datum. This percentile is then converted to a unit of standard deviation and the datum is plotted. Straight line array(s) through the data are then computed. When more than one line can be drawn through the data, the data are not homogeneous. Accordingly, if a maximum concentration for a specific substance is not among the data in the distribution considered to be representative of background levels, then that datum must be considered to be part of a distribution related to contamination.

According to our interpretation of the graphs, the latter situation is considered to be applicable to the maximum concentrations reported on the LLNL Livermore Site for antimony (Fig. C-1), arsenic (Fig. C-2), beryllium (Fig. C-4), cadmium (Fig. C-5), total chromium (Fig. C-6), cobalt (Fig. C-7), copper (Fig. C-8), lead (Fig. C-9), mercury (Fig. C-10), molybdenum (Fig. C-11), nickel (Fig. C-12), silver (Fig. C-14), zinc (Fig. C-17), cesium-137 (Fig. C-18), plutonium 239 + 240 (for values reported as "outliers" by Brekke *et al.*, 1989; see Fig. C-19), and tritium (for very high levels; see Fig. C-22). Similarly, according to our interpretation of the data plotted according to the procedure of Michels (1971) maximum concentrations detected on the LLNL Livermore Site of barium (Fig. C-3), selenium (Fig. C-13), thallium (Fig. C-15), vanadium (Fig. C-16), potassium-40 (Fig. C-20), thorium-232 (Fig. C-21), and uranium-238

(Fig. C-23) are considered to be possible background levels. Furthermore, none of the maximum concentrations of inorganic substances or radioactive chemicals presented in Figures C-1 through C-23 detected in surface soils sampled from the arroyos nearby the LLNL Livermore site is considered to be distinct from background.

Appendix C References

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Carpenter, D. W., A. L. Lamarre, N. B. Crow, and P. M. Swearengen (1988), *Closure Plan for the Decommissioned High Explosives Rinse-Water Lagoons at Lawrence Livermore National Laboratory Site 300*, Lawrence Livermore National Laboratory, Livermore, Calif. (UCID-21369), pp. A-39 to A-40 (Table A-32); and p. 33 (Fig. 14).

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Michels, D. E. (1971), "Log-Normal Analysis of Data for Plutonium in the Outdoors," in *Proceedings of Environmental Plutonium Symposium Held at LASL, August 4–5, 1971*, Los Alamos National Laboratory, Los Alamos, N.Mex. (LA-4756), pp. 105–111.

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Table C-1. Soil concentration (TTLC[a]) data for inorganic chemicals monitored at LLNL,[b] Site 300,[c] and reported for selected counties in California.[d]

Chemical	LLNL site location[e]	Maximum soil concentration at LLNL $C_{s\text{-}max}^{inorg}$ (mg/kg)	Maximum soil concentration at Site 300 (mg/kg)	Maximum soil concentration in arroyos adjacent to LLNL site	Selected counties for which soil concentrations (TTLCs) are reported						
					San Joaquin (mg/kg)	Butte (mg/kg)	Lassen (mg/kg)	Los Angeles (mg/kg)	Kern (mg/kg)	Fresno (mg/kg)	Shasta (mg/kg)
Antimony	SSS-009	13	<10	<LOD[f]	<2	<2	<2	<2	10	<10	<10
Arsenic	SSS-003	14	2.8	2.0	4.7	4	2.7	26	10	2.4	2.7
Barium	SSS-003	300	210	130	310	170	200	100	90	30	200
Beryllium	SSD-009	4	0.8	<LOD[g]	1.8	1.6	1.7	0.59	0.2	0.09	0.61
Cadmium	SSS-009	23	<0.2	1.0	1.4	1.1	1.1	1.8	0.72	0.44	2.8
Chromium (total)	SSS-009	1500	28	27	25	26	14	26	22	11	49
Cobalt	ALPE	22	12	11	10	7.7	15	3.9	6.3	4.3	18
Copper	SSS-009	530	26	2.1	15	10	14	53	33	3.7	27
Lead	SSD-009	320	20	20	68	52	45	100	41	20	47
Mercury	SSD-009	20	<0.01	<LOD[g]	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Molybdenum	SSS-009	16	6	<LOD[f]	1.4	<2	<2	1.4	<2	<2	<2
Nickel	SSS-009	670	34	32	22	26	15	5.3	5.8	4.1	18
Selenium	ALPW	0.2	<0.2	<LOD[f]	<0.2	<0.2	<0.2	<0.2	<0.2	0.2	7.8
Silver	SSS-009	7.4	<1.0	0.9	<0.6	<0.6	<0.6	<0.6	<0.8	<0.08	<0.08
Thallium	CDB	9	10	<LOD[f]	6.8	5	4	3	5.2	2	<2
Vanadium	SSS-003	40	78	26	43	19	23	11	18	14	51
Zinc	SSD-009	750	64	59	50	36	23	240	52	31	30

[a]The total threshold limit concentration (TTLC) is determined by nitric acid digestion (see DHS, 1984).

[b]Monitoring data for the LLNL site appear in Appendix N of the RI report (Thorpe *et al.*, 1990).

[c]Maximum TTLCs for soils at depths between 1.5 and 2.1 ft from the surface at two locations near Bldg. 827 (sampling locations B-827-C2 and B-827-C3) at LLNL Site 300. [See Table C-2 in this appendix and Table A-32 (pp. A-39 to A-40) in Carpenter *et al.*, 1988.]

[d]See California Energy Commission (1985).

[e]As noted in Section 3, the locations for samples SSS-009 and SSD-009 have been remediated.

[f]Limit of detection (LOD) is equal to 1.0 mg/kg for this chemical.

[g]Limit of detection (LOD) is equal to 0.1 mg/kg for this chemical.

Table C-2. Soil concentration (TTLC[a]) data for inorganic chemicals monitored at Site 300.[b]

Chemical	Near-surface soil concentrations at Site 300 Location: B-827-C2 Depth (ft): 1.6 to 2.1 (mg/kg)	 B-827-C3 1.5 to 1.8 (mg/kg)
Antimony	<10	<10
Arsenic	2.8	1.4
Barium	140	210
Beryllium	0.8	0.6
Cadmium	<0.2	<0.2
Chromium (total)	24	28
Cobalt	12	11
Copper	26	26
Lead	16	20
Mercury	<0.01	<0.01
Molybdenum	<2.0	6
Nickel	24	34
Selenium	<0.2	<0.2
Silver	<1.0	<1.0
Thallium	8	10
Vanadium	78	56
Zinc	64	52

[a]The TTLC is determined by nitric acid digestion (see DHS, 1984).

[b]TTLCs for soils at two locations near Bldg. 827 (sampling locations B-827-C2 and B-827-C3) at Site 300. [See Table A-32 (pp. A-39 to A-40) in Carpenter *et al.*, (1988).]

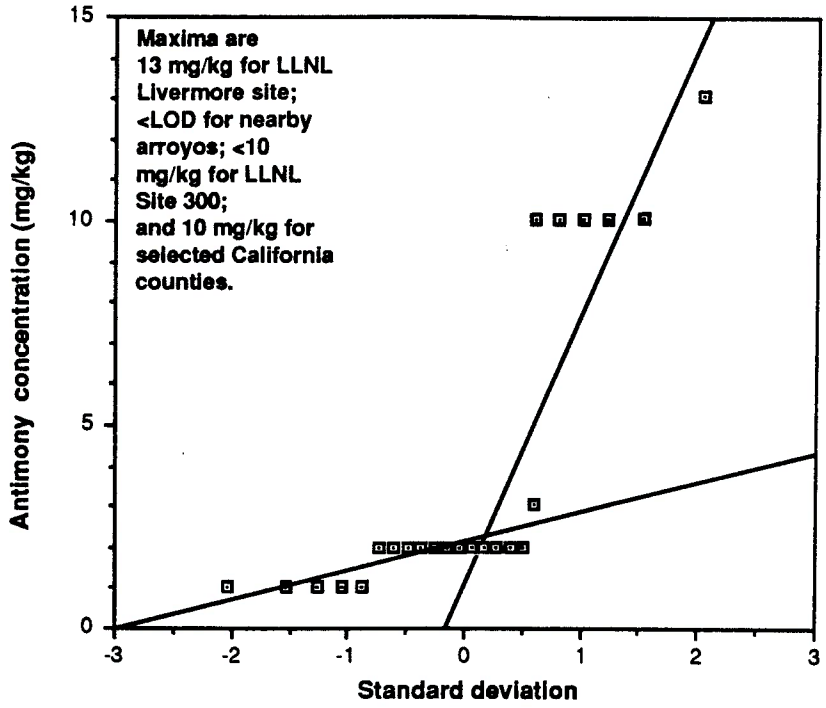

Figure C-1. Total threshold limit concentrations for antimony from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

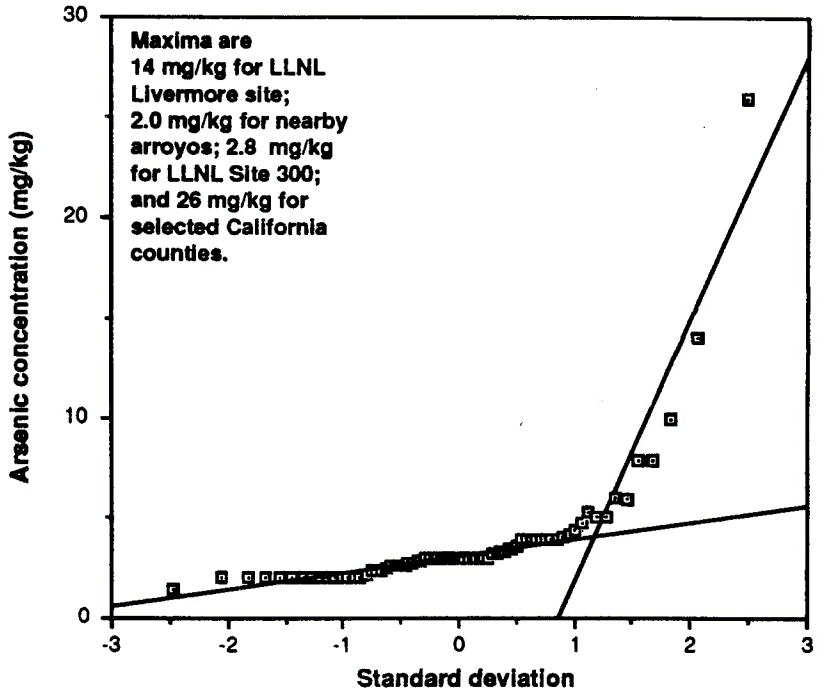

Figure C-2. Total threshold limit concentrations for arsenic from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

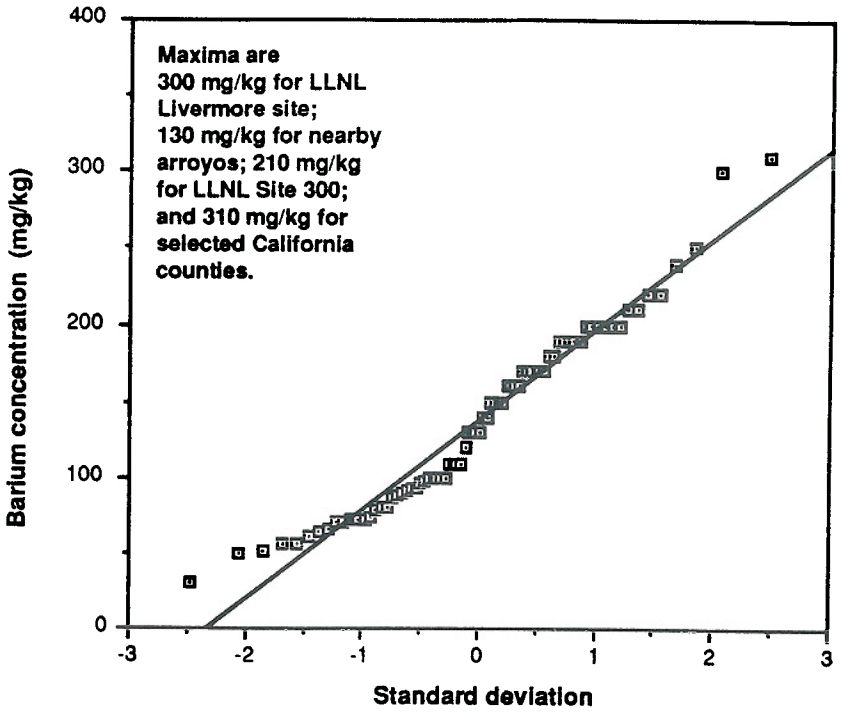

Figure C-3. Total threshold limit concentrations for barium from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

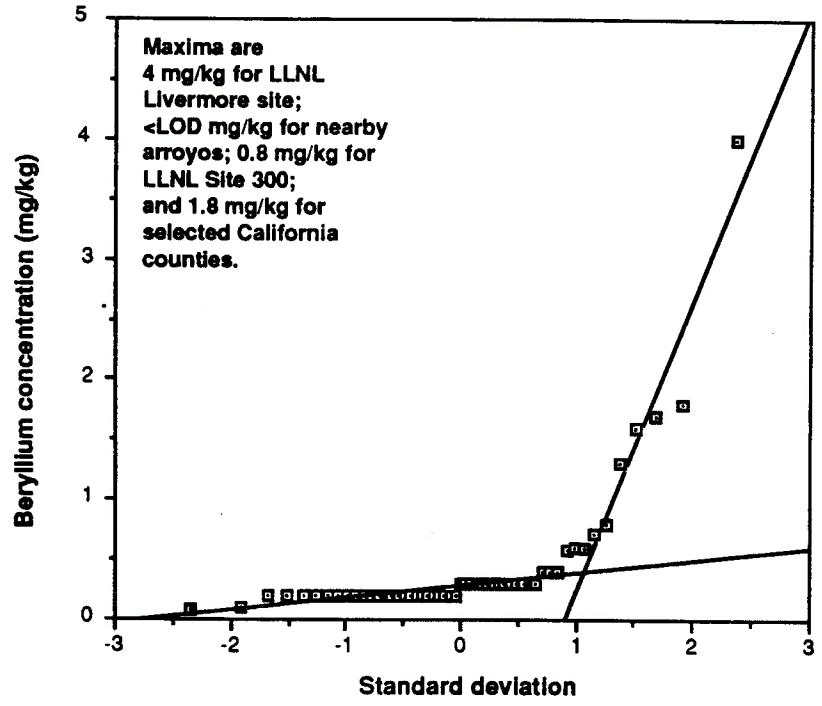

Figure C-4. Total threshold limit concentrations for beryllium from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

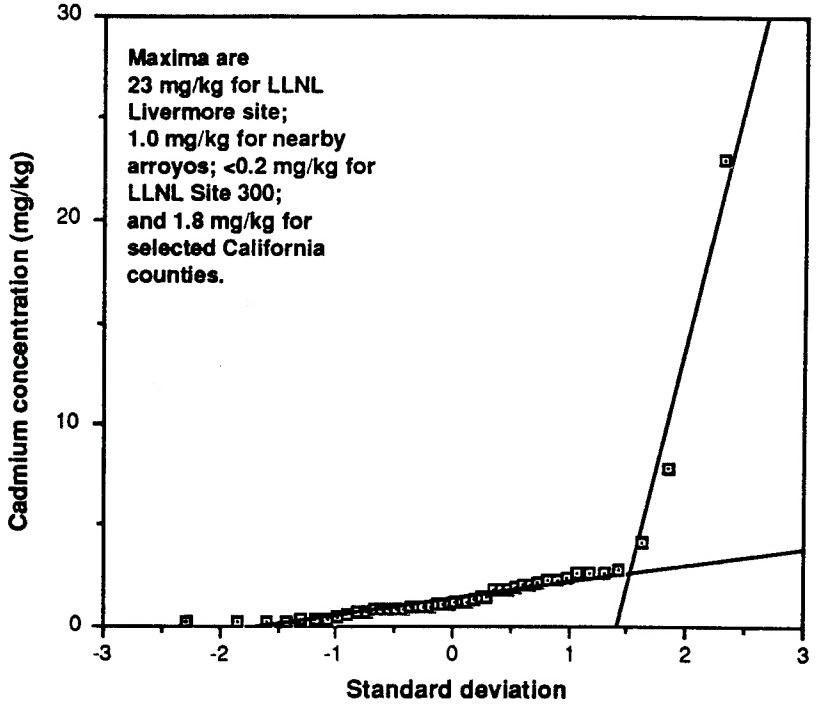

Figure C-5. Total threshold limit concentrations for cadmium from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

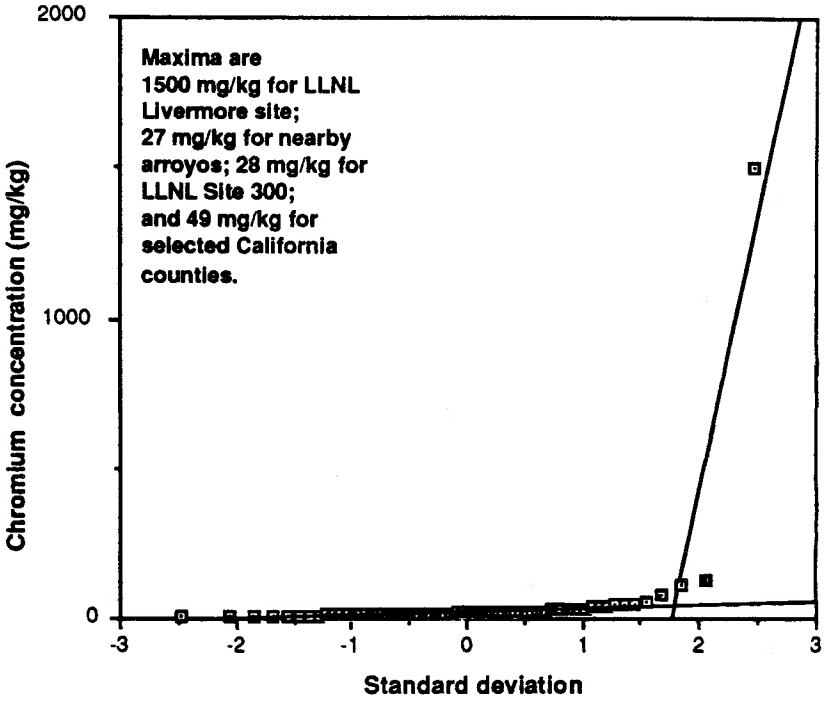

Figure C-6. Total threshold limit concentrations for total chromium from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

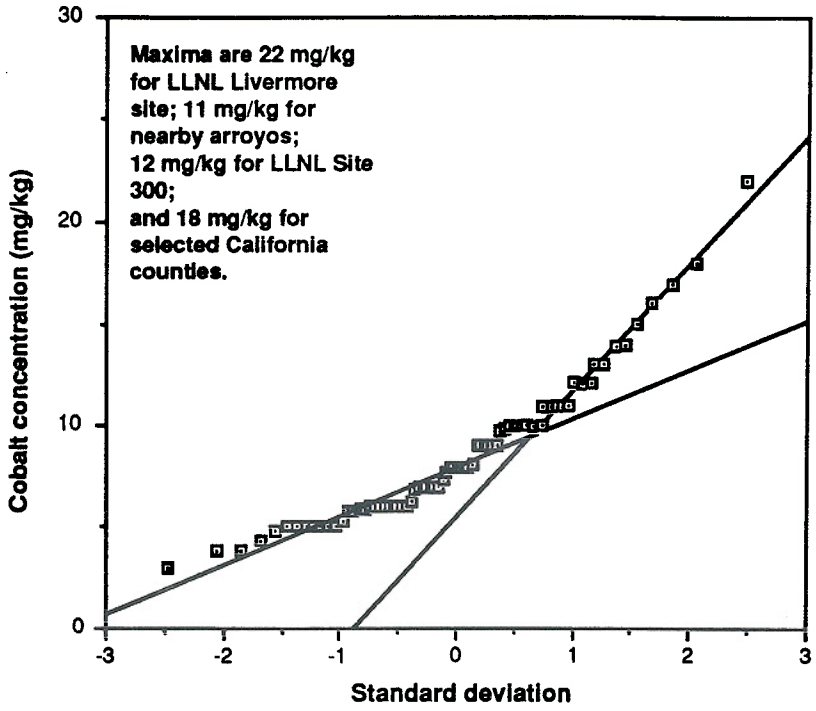

Figure C-7. Total threshold limit concentrations for cobalt from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

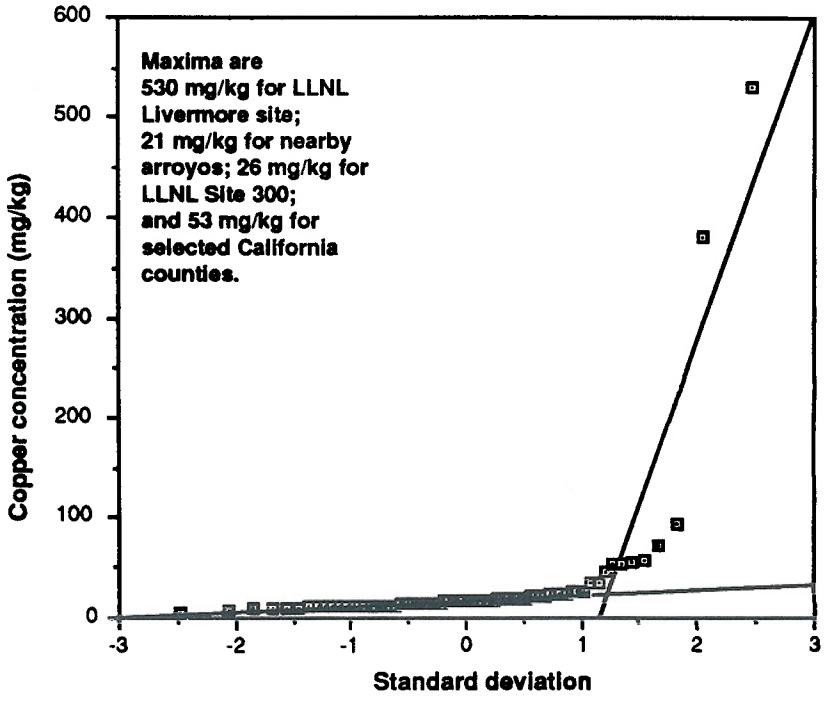

Figure C-8. Total threshold limit concentrations for copper from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

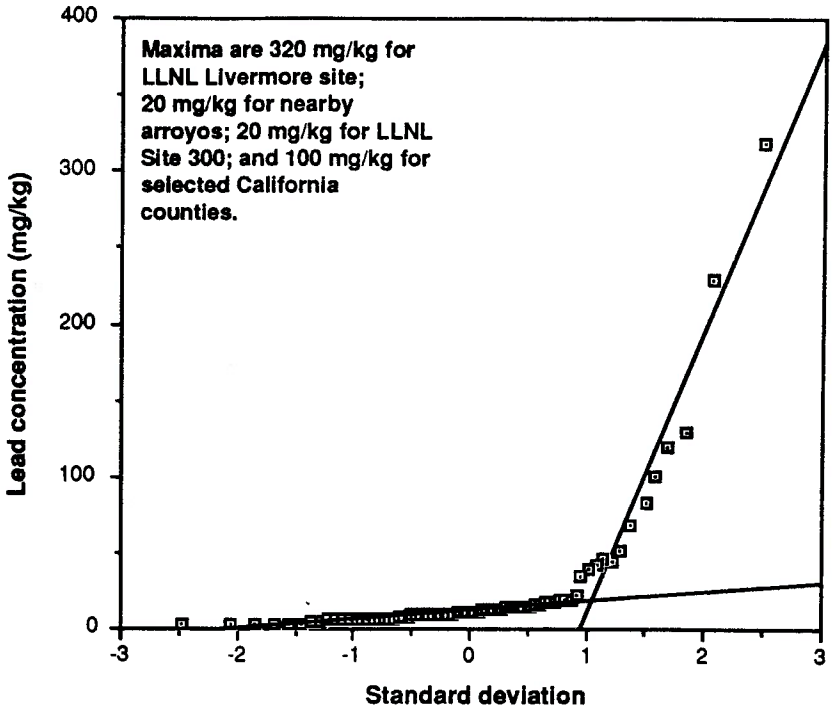

Figure C-9. Total threshold limit concentrations for lead from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

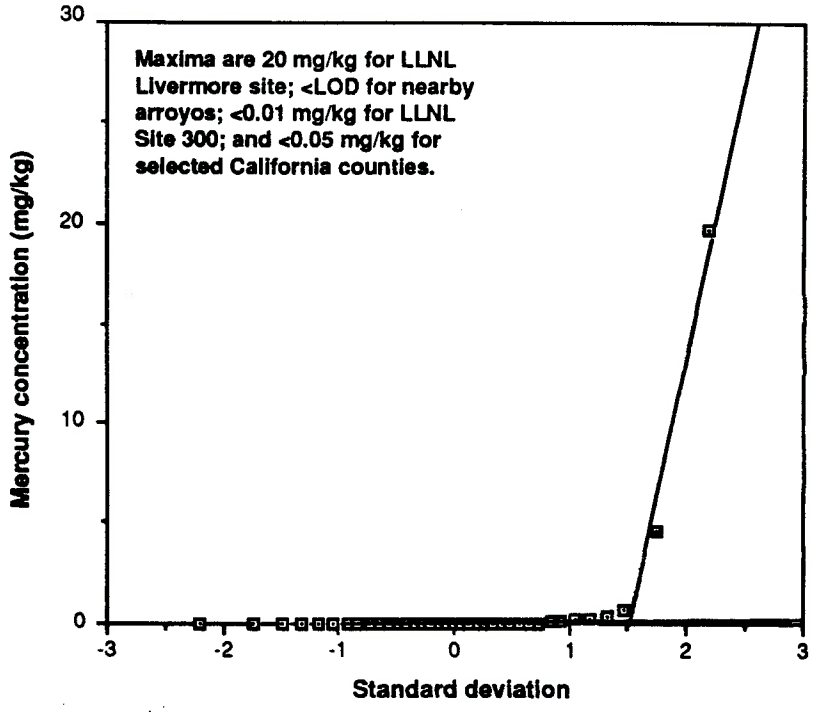

Figure C-10. Total threshold limit concentrations for mercury from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

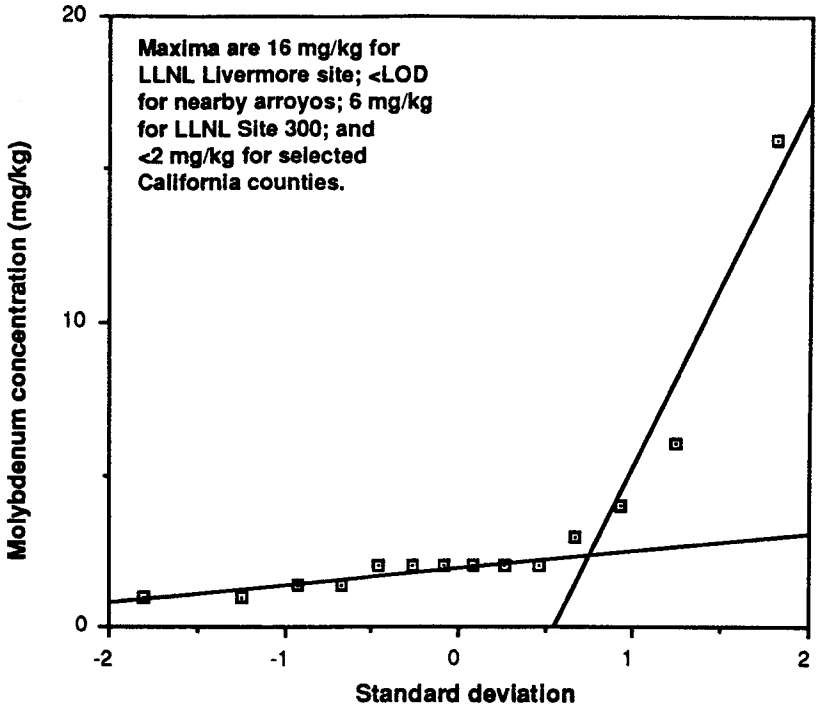

Figure C-11. Total threshold limit concentrations for molybdenum from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

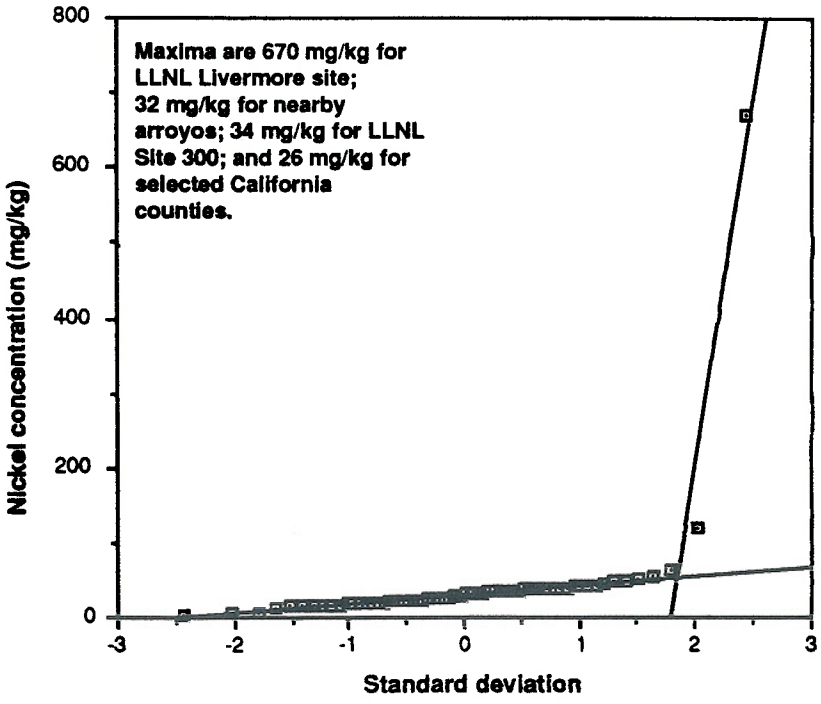

Figure C-12. Total threshold limit concentrations for nickel from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

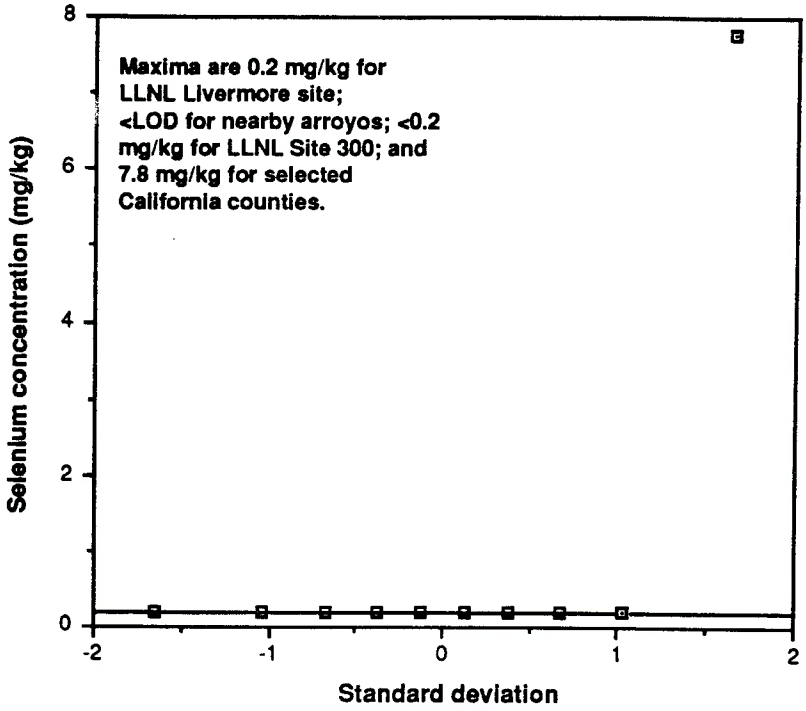

Figure C-13. Total threshold limit concentrations for selenium from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

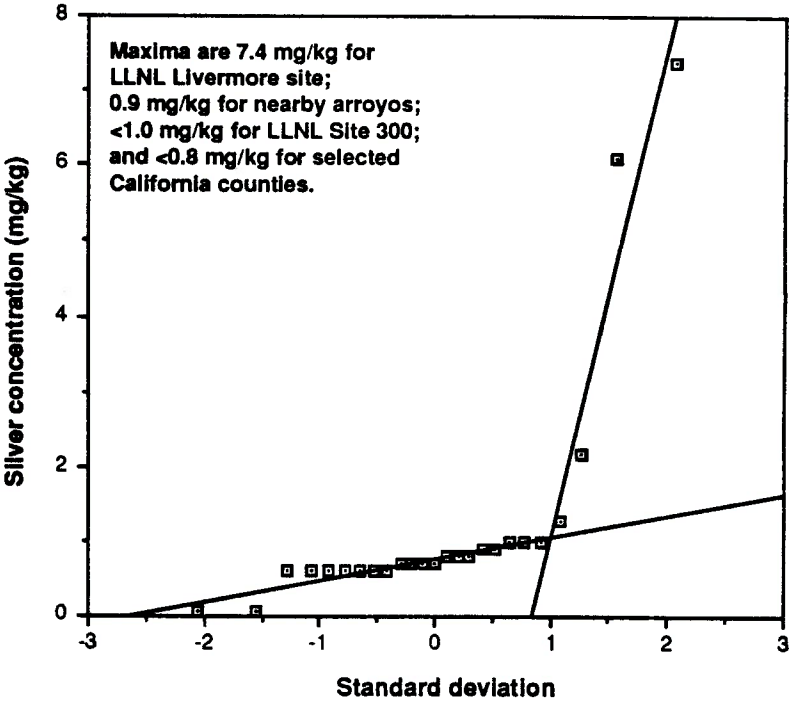

Figure C-14. Total threshold limit concentrations for silver from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

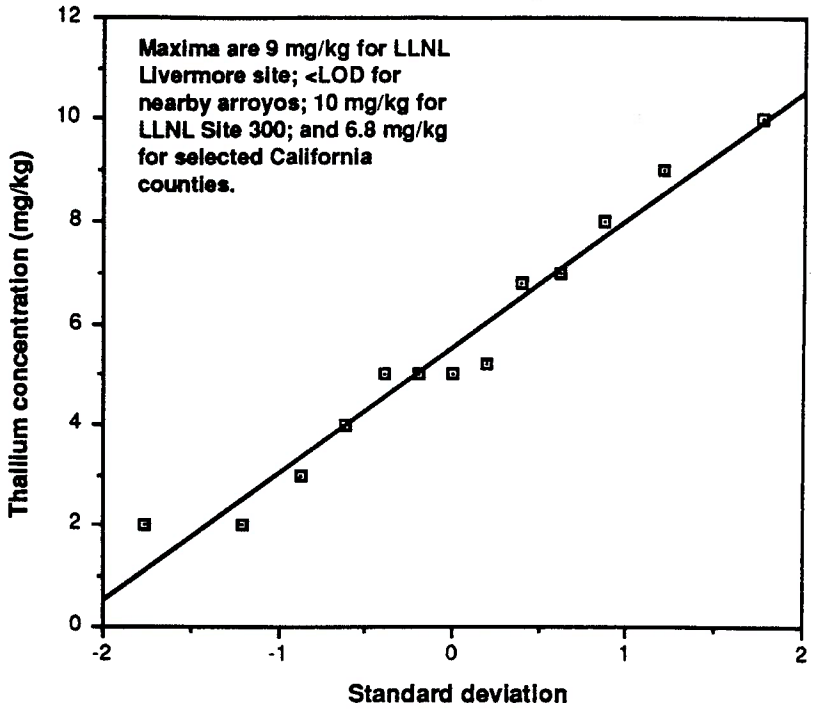

Figure C-15. Total threshold limit concentrations for thallium from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

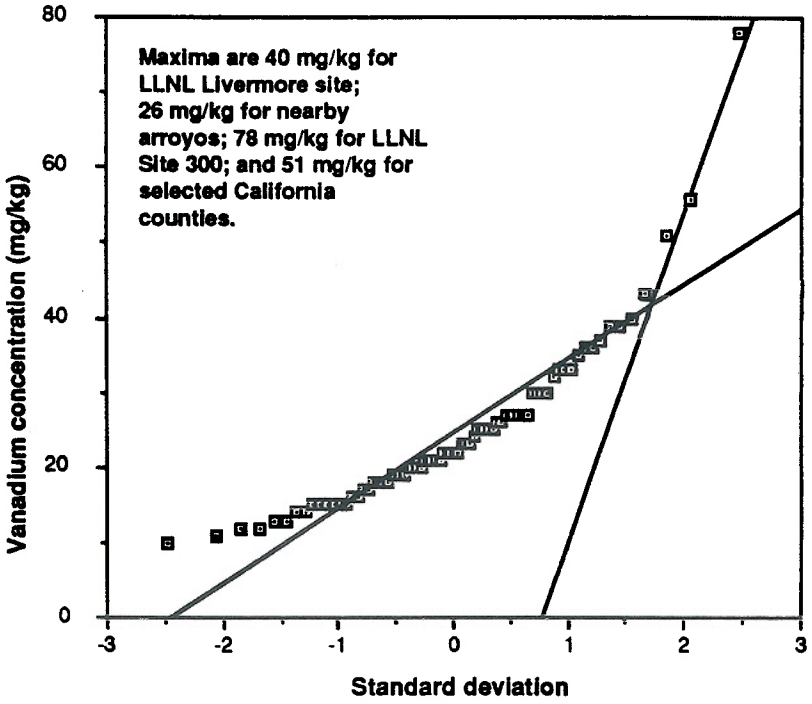

Figure C-16. Total threshold limit concentrations for vanadium from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

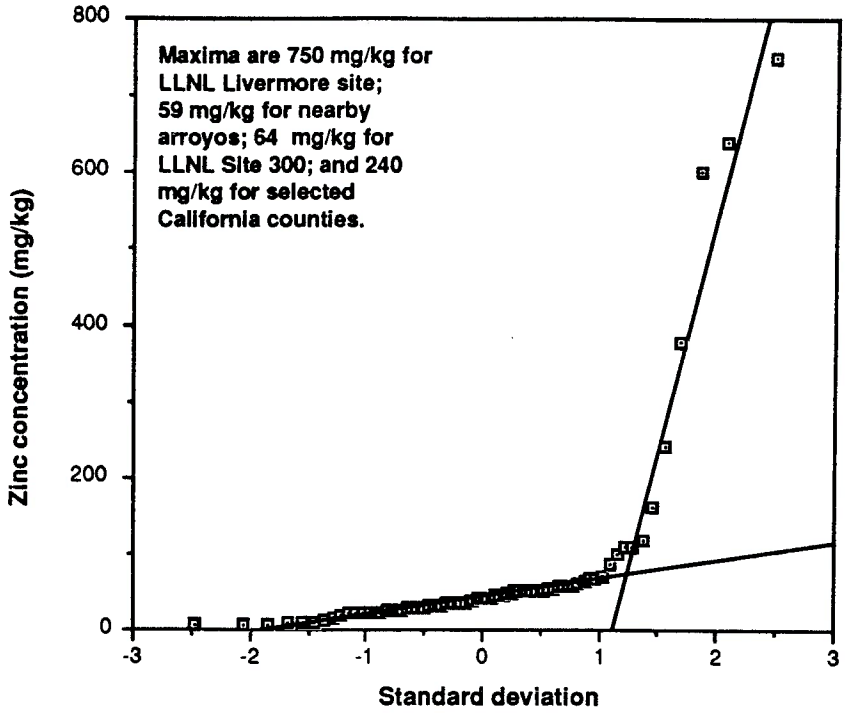

Figure C-17. Total threshold limit concentrations for zinc from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), LLNL Site 300, and selected California counties.

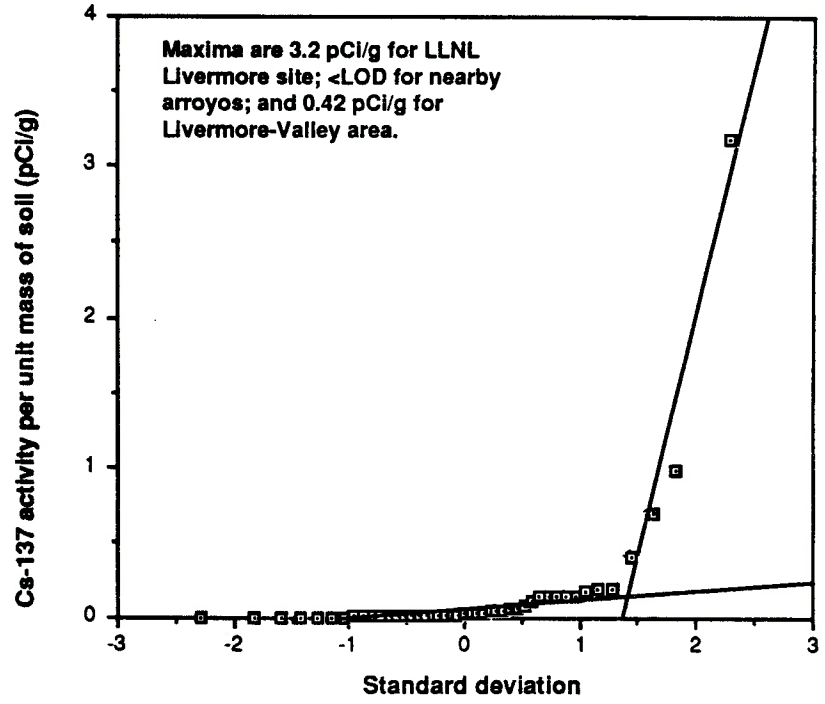

Figure C-18. Cesium-137 concentrations in pCi/g from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), and the Livermore-Valley area.

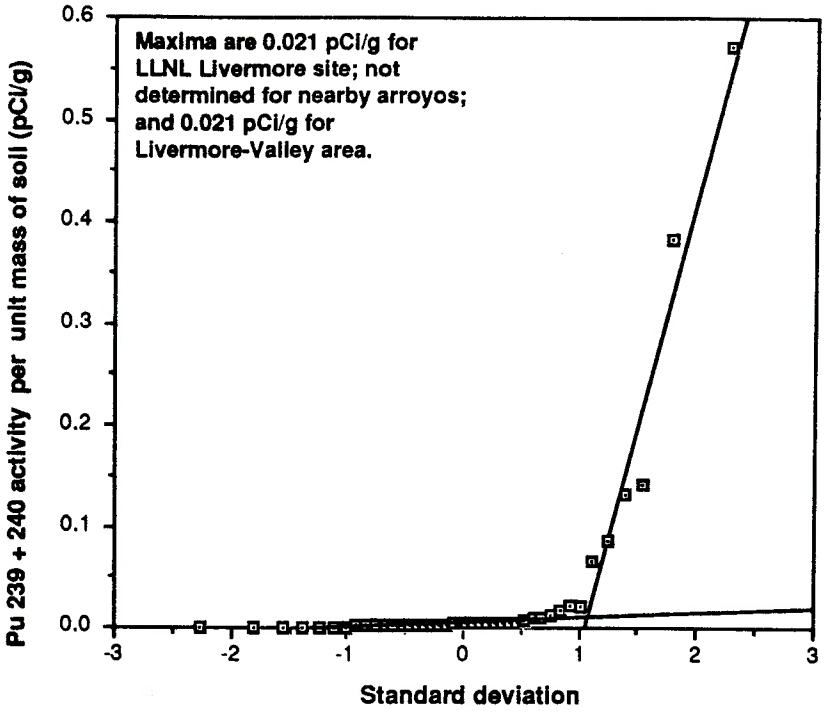

Figure C-19. Plutonium 239 + 240 concentrations in pCi/g from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), and the Livermore-Valley area (excluding data reported as outliers by Brekke *et al.*, 1989).

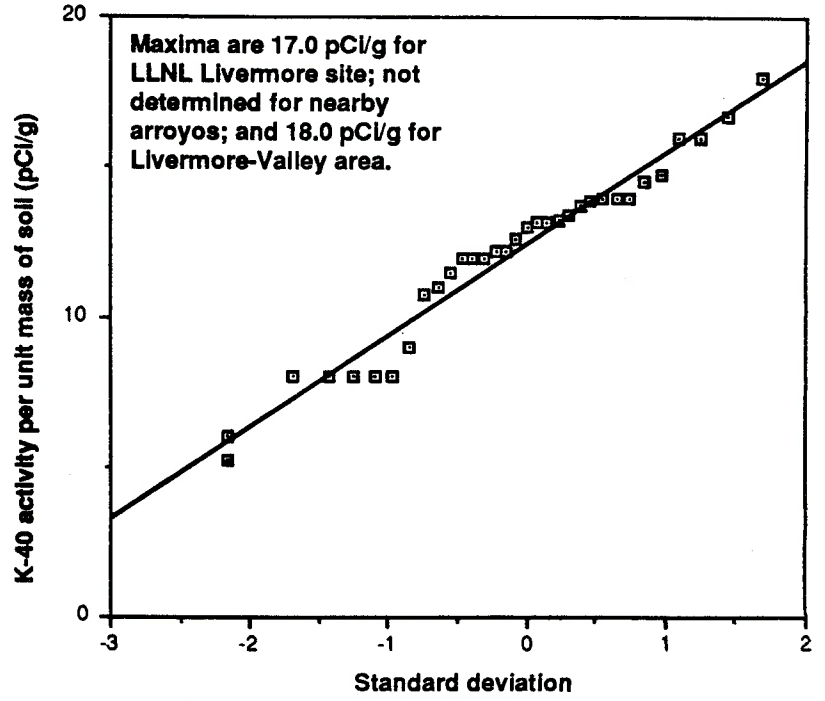

Figure C-20. Potassium-40 concentrations in pCi/g from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), and the Livermore-Valley area.

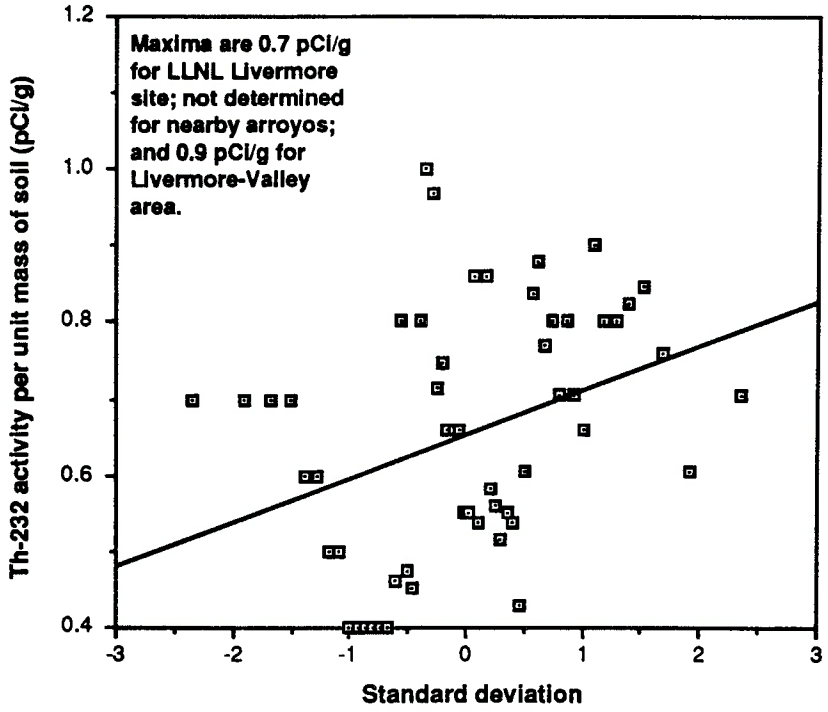

Figure C-21. Thorium-232 concentrations in pCi/g from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), and the Livermore-Valley area.

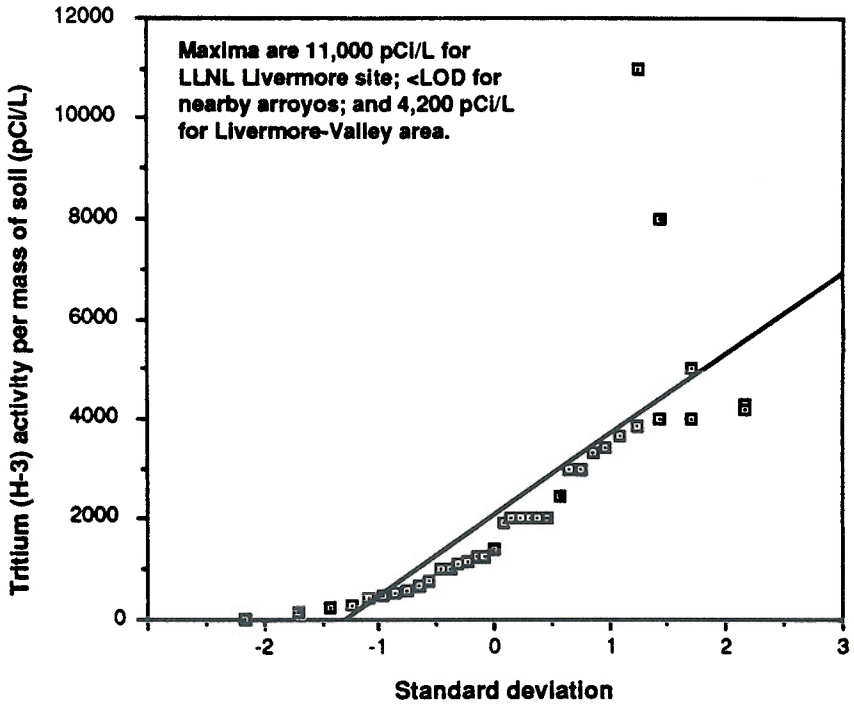

Figure C-22. Tritium (H-3) concentrations measured as activity in recovered water (pCi/L) from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), and the Livermore-Valley area (offsite on Sandia National Laboratories property, exclusively; see Brekke *et al.*, 1989).

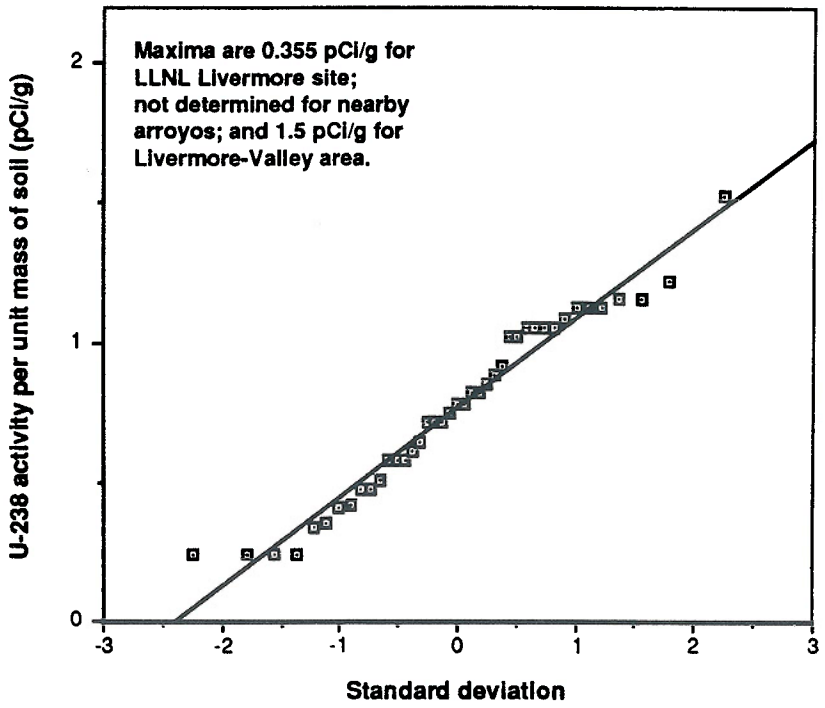

Figure C-23. Uranium-238 concentrations in pCi/g from soil monitoring data for the LLNL Livermore site and the arroyos nearby (above the limit of detection [LOD]), and the Livermore-Valley area.

Appendix D

Toxicity Assessment of VOCs

Appendix D

Toxicity Assessment of VOCs

L. C. Hall and K. T. Bogen

Each of the VOCs addressed in the Baseline Public Health Assessment has been the subject of extensive research. This appendix provides pertinent background data on their genotoxicity and carcinogenicity to laboratory animals and humans.

Trichloroethylene

Genotoxic Effects

Short-term genotoxicity testing of TCE has produced equivocal results. In general, TCE has given negative results in bacterial assays of mutagenicity, although Greim *et al.* (1975) and Simmon *et al.* (1977) reported weakly positive results in *Escherchia coli* and *Salmonella typhimurium*, respectively, in the presence of exogenous metabolic activation. However, Greim *et al.* did not specify the purity of the TCE used, and Henschler (1977) demonstrated that two common stabilizers of TCE were directly mutagenic to *S. typhimurium.*

TCE has induced mutations, gene conversions, and mitotic recombination in the yeast *Saccharomyces cerevisiae* when metabolic activation has been added (Bronzetti *et al.*, 1978; Callen *et al.*, 1980). A single study (Duprat and Gradiski, 1980) reported that TCE induced chromosomal aberrations in rodents.

Two studies performed with humans occupationally exposed to TCE reported an increased incidence of sister chromatid exchange (Gu *et al.*, 1981) and of hypodiploid cells (Konietzko *et al.*, 1978) in circulating lymphocytes. Both studies were seriously flawed, however, and do not prove clastogenic activity. Beliles *et al.* (1980) observed a slight increase in unscheduled DNA synthesis in cultured human cells, but similar experiments with rodent hepatocytes gave negative results. Several studies suggest TCE (or a metabolite) can bind to DNA (Miller and Guengerich, 1983; Bergman, 1983; Stott *et al.*, 1982).

In general, the results of short-term tests indicate that TCE is weakly mutagenic. Microsomal activation tests indicate that one or more metabolites of TCE may be the causative agent. Comprehensive reviews of TCE's genotoxicity are available in U.S. EPA (1985a) and Bogen *et al.* (1988).

Carcinogenicity in Animals

The carcinogenicity of TCE was evaluated in 11 separate animal bioassays. Not all of these studies were conducted with equal scientific rigor, nor have all yielded results of equal scientific

significance. Because of the size of this data base, the following discussion focuses only on the most significant results of each study. A comprehensive review of TCE's carcinogenicity can be found in U.S. EPA (1985a) and Bogen *et al.* (1988). A summary of relevant information is available in U.S. EPA (1988a).

In 1976, the National Cancer Institute (NCI, 1976a) released the results of the first lifetime study of the carcinogenicity of TCE to animals. This report indicated that oral administration of industrial-grade TCE 5 d/wk for 78 wk to B6C3F1 mice induced a significant increase in the incidence of hepatocellular carcinoma in mice. Male mice of both dosage groups [time-weighted average (TWA) doses of 1,169 mg/kg or 2,339 mg/kg] and high-dose females (TWA dose of 1,739 mg/kg) developed hepatocellular carcinomas in significantly greater numbers than control animals (Appendix Table D-1). In seven animals (four low-dose and three high-dose males), these cancers metastasized to the lung. Rats in this study received either 549 mg/kg (males) or 1,097 mg/kg (females) of TCE by the same treatment protocol as mice. In male rats, administration of TCE was associated with significantly ($p = 0.001$) lower survival rates than were observed in untreated controls. Histopathological examination of animals found no evidence that TCE significantly affected the incidence of specific or total tumors. In an analysis of this study, the International Agency for Research on Cancer (IARC, 1982) concluded that the substantial rate of early mortality in rats rendered that portion of the NCI study inadequate for an evaluation of the carcinogenicity of TCE. Various individuals and groups have also questioned the validity of the results in mice on the basis that the TCE was contaminated with epichlorohydrin (ECH), a known mutagen, as well as small quantities of other potentially toxic substances (U.S. EPA, 1985a).

To resolve the issue of whether these contaminants contributed to the carcinogenicity of TCE in mice, the National Toxicology Program (NTP) conducted another lifetime bioassay of TCE with B6C3F1 mice and F344/N rats (NTP, 1983). The TCE used in this study was free of ECH and was administered by gavage 5 d/wk for 103 wk. Rats received 500 or 1,000 mg/kg of TCE; mice received 1,000 mg/kg. As in the NCI (1976a) study, a significantly higher incidence of hepatocellular carcinoma was induced in male and female mice by treatment with TCE (Appendix Tables D-1 and D-2). Compared to the incidence in controls, female mice dosed with TCE also had a significantly ($p < 0.05$) increased incidence of hepatocellular adenomas. The unadjusted incidence of renal tubular-cell adenocarcinomas was identical in high-dose male rats and controls.

However, statistical tests that took survival differences into account showed that the incidence of these lesions was significantly ($p = 0.028$) higher in high-dose male rats that survived until the end of the experiment (3/16) compared to survivors from the control group (0/33). Toxic nephrosis significantly reduced the survival of treated rats to such an extent that the NTP (1983) considered the results "inadequate to evaluate the presence or absence of a carcinogenic response" in rats.

Industrial Bio-Test Laboratories conducted a lifetime inhalation bioassay of TCE in Osborne-Mendel rats and B6C3F1 mice. Study results were audited by the Manufacturing Chemists Association (MCA) and reported in Bell *et al.* (1978, as cited in U.S. EPA, 1985a). The experimental protocol consisted of exposing animals to 100, 300, or 600 ppmv of TCE 6 h/d, 5 d/wk for 104 wk. Male mice from all exposure groups developed significantly high incidences of hepatocellular carcinoma compared to controls (see Appendix Table D-3). Female mice

exposed to 600 ppmv showed a significant ($p < 0.05$) increase in the combined incidence of hepatocellular adenoma and hepatocellular carcinoma relative to controls. Tumor incidence in rats was not affected by exposure to TCE. However, the MCA audit found substantial deficiencies in the conduct of the study, as well as in the analysis of study results. The U.S. EPA (1985a) concluded that these deficiencies compromised the study results, thereby limiting their usefulness.

The latest NTP bioassay of TCE utilized ACI, August, Marshall, and Osborne-Mendel rats (NTP, 1988). Male and female rats of all four strains received 500 or 1,000 mg/kg of TCE 5 d/wk for 103 wk. Low-dose Osborne-Mendel rats developed a significantly ($p = 0.007$) higher incidence of renal tubular-cell adenomas. High-dose male Marshall rats developed a significantly ($p = 0.002$) higher incidence of testicular interstitial cell tumors, relative to controls (Appendix Table D-4). However, the incidence of testicular tumors was also high in untreated ACI rats, and TCE-treated ACI rats of both dosage groups had a lower incidence of these tumors than the controls. Consequently, it is difficult to evaluate the biological significance of this particular tumor. An audit of this study determined that documentation of animal breeding, animal identity, clinical observations, environmental conditions, and analytical chemistry data were inadequate to support any meaningful interpretation of the reported tumor incidence data. Nonetheless, the NTP Peer Review Panel made a point of noting the positive results obtained in this study, despite its limitations.

Van Duuren *et al.* (1979) used three separate protocols to examine the carcinogenicity of TCE. Mice were exposed to TCE by gavage or subcutaneous injection (0.5 mg once a week for 89 wk), or by dermal application. In the latter procedure, animals received a single application of 1.0 mg TCE followed 2 wk later by repeated applications of phorbol myristate acetate. Another group received dermal applications of 1.0 mg TCE three times a week for 83 wk. Regardless of the exposure protocol, no significant positive tumorigenic response was observed (Appendix Table D-5).

Henschler *et al.* (1980) exposed groups of mice, rats, and hamsters to 100 or 500 ppmv of stabilized TCE 6 h/d, 5 d/wk for 78 wk. Mice and hamsters were sacrificed 52 wk after the end of treatment, and rats, 78 wk after. Compared to controls, treated female mice developed a significantly ($p < 0.05$) elevated incidence of malignant lymphoma (Appendix Tables D-3 and D-4). Tumor incidence in all other groups of animals was not affected by treatment with TCE.

ICR mice exposed to 150 or 450 ppmv TCE 7 h/d, 5 d/wk for 104 wk developed pulmonary adenocarcinomas in significantly ($p < 0.05$) higher numbers than controls (Fukuda *et al.*, 1983). However, analysis of the combined incidence of pulmonary adenomas and adenocarcinomas in exposed mice revealed no significant differences from the incidence of these tumors in untreated controls (Appendix Table D-3). The tumor incidence in mice exposed to 50 ppmv TCE and in rats exposed to 50, 150, or 450 ppmv was not significantly affected by treatment (Appendix Table D-4).

Because of the controversy surrounding the use of ECH-contaminated TCE by the NCI (1976a), Henschler *et al.* (1984) tested the carcinogenicity of different samples of TCE, with or without ECH or 1,2-epoxybutane, also a suspected carcinogen. Bogen *et al.* (1988) calculated that male mice received TWA doses of 1,900 mg/kg and female mice, 1,400 mg/kg, 5 d/wk over an 18-month period. Administration of TCE with ECH alone or TCE with ECH and 1,2-epoxybutane was associated with a significant increase in forestomach papillomas or

carcinomas in male and female mice (Appendix Table D-1). Administration of TCE with 1,2-epoxybutane alone induced a significant ($p < 0.05$) increase in the incidence of squamous cell carcinomas in male mice relative to controls. Purified, amine-stabilized TCE did not significantly affect tumor incidence in dosed mice.

Wester *et al.* (1985) administered a mixture of 11 volatile organic compounds to rats in their drinking water. Solutions contained equal amounts of each compound, and were prepared by adding 0.22, 2.2, or 22 mg (total) of a mixture to 1 mL ethanol for each liter of water. Bogen *et al.* (1988) estimated that a rat in the highest dose group received approximately 0.2 mg/kg-d over the 25-month exposure period. Although no significant differences in tumor incidence were observed between treated and control animals, the administered doses may have been too small to cause tumors. Interpretation of these results is also complicated by the simultaneous administration of 11 separate compounds.

Between 1976 and 1983, Maltoni *et al.* (1986) conducted a comprehensive series of experiments with mice and rats to evaluate the carcinogenicity of TCE. In the only experiment in which TCE was administered by gavage (designated BT 301), Sprague-Dawley rats were given 50 or 250 mg/kg of TCE 4 or 5 d/wk for 52 wk. An apparent dose-related increase in leukemia was observed in treated males, but the incidence was not significantly different from controls (Appendix Table D-2).

Two 8-wk inhalation experiments with Sprague-Dawley rats (BT 302) and Swiss mice (BT 303) also gave negative results (Appendix Tables D-3 and D-4). However, exposure of Sprague-Dawley rats to TCE at concentrations of 100, 300, or 600 ppmv, 7 h/d, 5 d/wk for 104 wk (BT 304) resulted in a statistically significant increase in the incidence of Leydig cell tumors of the testes (Appendix Table D-4). With a single exception, these tumors were classified as benign. Five rats from the 600-ppmv group developed renal adenocarcinomas, a finding that was not statistically significant, but was deemed biologically significant because of the rarity of this tumor type.

Maltoni *et al.* (1986) used the same three exposure concentrations and the same dosing protocol to study TCE's carcinogenicity to Swiss and B6C3F1 mice (experiments BT 305 and BT 306/306 bis, respectively). (See Appendix Table D-3.) In these experiments, however, exposure lasted only 78 wk. In Swiss mice, the two highest concentrations of TCE induced a significant increase in the incidence of pulmonary tumors in male mice, compared to controls. High-dose males also had a higher incidence ($p < 0.05$) of hepatomas than controls. Tumor incidence in females was not affected by exposure to TCE. In female B6C3F1 mice from all three exposure groups, the total number of malignant tumors was significantly higher than controls. An increase in the incidence of pulmonary tumors was also significant ($p < 0.05$) in females exposed to 600 ppmv TCE. Tumor incidence in male mice was not affected by exposure to TCE.

It is generally accepted that the toxicity and carcinogenicity of TCE is due to the formation of one or more reactive metabolites (Buben and O'Flaherty, 1985; U.S. EPA, 1985a; Bogen *et al.*, 1988). To examine this hypothesis, Herren-Freund *et al.* (1987) administered either TCE or the TCE metabolites trichloroacetic acid (TCA) or dichloroacetic acid (DCA) to B6C3F1 mice in drinking water. Administration of TCE (3 or 40 mg/L) with or without prior initiation with ethylnitrosourea (ENU) did not affect the incidence of hepatocellular adenomas or hepatocellular carcinomas in treated animals, compared to controls (Appendix Table D-5). However both

metabolites, administered alone or after initiation with ENU, induced a significant ($p < 0.01$) increase in the incidence of both types of tumors (Appendix Table D-6).

Bogen *et al.* (1988) calculated a range of cancer potencies for metabolized TCE on the basis of tumor-incidence data from NCI (1976a), NTP (1983), Bell *et al.* (1978), Fukuda *et al.* (1983), and Maltoni *et al.* (1986). These values, and the sets of data on which they are based, are listed in Appendix Table D-7. The median cancer potency of metabolized TCE used for our health risk assessment, 0.017 $(mg/kg\text{-}d)^{-1}$ (Table 5-13), was calculated from the range of potencies listed in Appendix Table D-7.

Carcinogenicity in Humans

Of the three retrospective studies available of human occupational exposure to TCE, only Hardell *et al.* (1981) found a statistically significant ($p < 0.05$, Chi-square test) association between exposure to TCE and an elevated risk of cancer (malignant lymphoma). However, the method of data collection and other flaws in study design limit the usefulness of these data in evaluating TCE's carcinogenicity to humans. Paddle (1983) and Novotna *et al.* (1979) found no evidence that occupational exposure to TCE increased the risk of liver cancer.

An historical cohort study of Swedish men occupationally exposed to TCE found no excess mortality due to cancer (Axelson *et al.*, 1978). However, the size of the cohort was relatively small, and the duration of TCE exposure was poorly defined. Both of these factors led the U.S. EPA (1985a) to conclude that there were severe limitations in overall study design and interpretation. Malek *et al.* (1979, as cited in U.S. EPA, 1985a) observed six cases of cancer among 57 dry cleaners who had used TCE as a cleaning solvent for 1 y. The period of time that had elapsed since exposure to TCE ranged from 5 to 50 y. Statistical analysis revealed that the observed incidence of cancer in this group of men was not significantly different from that expected. Tola *et al.* (1980) reported the first results of an ongoing cohort study of Finnish workers exposed to TCE. Of the approximately 2,000 individuals included in this study, no significant differences were noted between the number of observed and expected deaths from cancer. However, the short follow-up period (6 to 13 y) may have limited the ability of this study to detect an effect of TCE exposure.

Tetrachlorethylene

Genotoxic Effects

Little evidence exists that commercial and technical-grade preparations of PCE are weakly mutagenic. Cerna and Kypenova (1977) reported in an abstract that PCE of unspecified purity induced mutations in *Salmonella typhimurium*. The same authors also reported that PCE was mutagenic in a host-mediated assay. These results have not been corroborated in data published by Greim *et al.* (1975), Bartsch *et al.* (1979), or Kringstad *et al.* (1981). Furthermore, a comprehensive series of Ames tests conducted by the Environmental Mutagenesis Test Development Program for the NTP (1986) concluded that PCE was not mutagenic to any of the four strains of *S. typhimurium* tested.

Callen *et al.* (1980) found that PCE (purity not given) induced substantial increases in the frequency of mitotic recombination and gene conversion in *Saccharomyces cerevisiae*. However, Bronzetti *et al.* (1983) obtained only negative results when they studied the effect of PCE on the same species of yeast. PCE was inactive in short-term tests with *Drosophila* and mouse lymphoma cells. No evidence of cytogenetic damage (chromosome aberrations or sister chromatid exchange) was obtained when Chinese hamster ovary cells were incubated with PCE.

When PCE was tested for its ability to induce unscheduled DNA synthesis, conventional liquid-phase exposure protocols gave either questionable (Beliles *et al.*, 1980) or clearly negative results (Williams, 1983; Williams and Shimada, 1983, as cited in U.S. EPA 1985b). However, vapor-phase exposure of hepatocytes to stabilized and "low-stabilized" PCE reportedly induced an increase in unscheduled DNA synthesis (Williams and Shimada, 1983).

A concentration-dependent mutagenic response was observed when a metabolite of PCE, tetrachloroethylene oxide (PCE oxide), was incubated with *S. typhimurium*. Evidence of genetic toxicity was also observed with *Escherchia coli* (strain polA1$^-$), but PCE oxide was not mutagenic to *E. coli* (strain WP2 uvrA) (Kline *et al.*, 1982). Two other metabolites of PCE, TCA and trichloroethanol, were inactive when tested with *S. typhimurium*. Evidence suggests that trichloroethanol may induce sister chromatid exchange in cultured human lymphocytes (Gu *et al.*, 1981).

Carcinogenicity in Animals

The National Cancer Institute (NCI, 1977) examined the carcinogenicity of PCE administered by gavage to B6C3F1 mice and Osborne-Mendel rats. In this study, the TWA daily doses of PCE were 536 or 1,072 mg/kg for male mice, 386 or 722 mg/kg for female mice, 471 or 941 mg/kg for male rats, and 474 or 949 mg/kg for female rats. Administration of PCE in corn oil 5 d/wk for 78 wk was associated with a statistically significant ($p < 0.001$) increase in the incidence of hepatocellular carcinomas in male and female mice in both dosage groups (Appendix Table D-8). Treated animals also developed a high incidence of toxic nephropathy, which was not observed in controls. The median survival time of male and female mice decreased with increasing dose of PCE.

The survival of rats was also adversely affected by exposure to PCE. Approximately half of the high-dose males had died by the 44th week of treatment and half of the high-dose females died by the 66th week. The NCI (1977) determined that there was a significant ($p < 0.001$) association between treatment with PCE and increased mortality in rats. The excessive mortality in rats dosed with PCE precluded use of data from this portion of the study in evaluating PCE's potential carcinogenicity.

Subsequently, the National Toxicology Program (NTP) sponsored a bioassay in which B6C3F1 mice and F344/N rats were exposed to PCE by inhalation, 6 h/d, 5 d/wk for 103 wk (NTP, 1986). Mice were exposed to 0, 100, or 200 ppmv; rats were exposed to 0, 200, or 400 ppmv PCE. Male mice from both exposure groups and high-dose female mice had significantly ($p < 0.001$) lower survival rates than untreated controls. The incidence of hepatocellular carcinoma was significantly increased among treated mice of both sexes from both exposure groups (Appendix Table D-8). The number of hepatocellular adenomas was also

increased in treated animals compared to controls, but the increase was not statistically significant.

Exposure to PCE adversely affected survival among male rats, but had little effect on treated females. Inhalation of PCE was also associated with a significant increase in the incidence of mononuclear cell leukemia in rats of both sexes (Appendix Table D-8). An increased incidence of renal tubular-cell adenomas and adenocarcinomas was documented in male rats from both exposure groups, but not in females. The incidence of these tumors appeared to be dose related, but was not significantly different from control levels.

Two other studies have examined the carcinogenicity of PCE to animals. Rampy *et al.* (1978) exposed rats to 300 or 600 ppmv PCE by inhalation, 6 h/d, 5 d/wk for 12 months. Although no evidence of carcinogenicity was found, interpretation of this study is open to question due to the less-than-lifetime duration of treatment. Theiss *et al.* (1977) injected mice intraperitoneally with PCE 3 times a week for 4 to 16 wk. Upon sacrifice 24 wk later, animals were examined for the presence of pulmonary tumors. This test has not produced positive results with several known animal carcinogens. The fact that there was no evidence that PCE induced pulmonary tumors has little significance in the evaluation of PCE's carcinogenic potential.

Bogen *et al.* (1987) calculated a series of cancer potency values for metabolized PCE based on the tumor-incidence data from NCI (1977) and NTP (1986). These values and supporting data are provided in Appendix Table D-8. The median cancer potency of metabolized PCE (see Table 5-13), used for our health risk assessment, 0.27 $(mg/kg\text{-}d)^{-1}$, was calculated from the range of potencies listed in Appendix Table D-8.

Carcinogenicity in Humans

Chronic occupational exposure to PCE has been associated with an excess risk of cancer of the kidney, bladder, cervix, colon, and respiratory system. However, the six epidemiological studies completed to date were compromised by the investigator's inability to define the extent of exposure to PCE, by exposure of the cohort to other (potentially carcinogenic) solvents, and by failure to control for smoking and the socioeconomic status of the cohort (Blair *et al.*, 1979; Katz and Jowett, 1981; Kaplan, 1980; Lin and Kessler, 1981; Duh and Asal, 1984; Brown and Kaplan, 1987). In-depth reviews of these studies can be found in IARC (1982), Reichert (1983), U.S. EPA (1985b), and Bogen *et al.* (1987). In general, the equivocal results of these studies add only limited information to our knowledge of the health hazards associated with exposure to PCE and do not provide any direct evidence that PCE is carcinogenic to humans.

Chloroform

Genotoxic Effects

The genotoxicity of chloroform was evaluated in a broad spectrum of short-term tests, in both prokaryotic and eukaryotic organisms. In the Ames test with *Salmonella typhimurium*, chloroform was studied in five separate tester strains and in three exposure protocols (suspension, plate-incorporation, and vapor-phase). Negative results were obtained in all

instances (Uehleke *et al.*, 1977; Simmon *et al.*, 1977; Van Abbe *et al.*, 1982; Gocke *et al.*, 1981). Chloroform reportedly gave a positive response in *S. typhimurium* strain TA 1537 in a host-mediated assay with mice (Agustin and Lim-Sylianco, 1978). However, data discrepancies and omission of the exposure concentration(s) make it difficult to evaluate the significance of this report. Kirkland *et al.* (1981) found no evidence that chloroform was mutagenic to *Escherichia coli.*

Chloroform did not induce mutations at the hypoxanthine-guanine phosphororibosyl transferase (HGPRT) locus in cultured lung fibroblasts (Sturrock, 1977). Tests for the initiation of hepatocellular foci were also negative (Pereira *et al.*, 1982; Deml and Oesterle, 1985, 1987). Two studies reported a slight but nonsignificant increase in the number of micronuclei in erythrocytes from mice exposed to chloroform (Agustin and Lim-Sylianco, 1978; Gocke *et al.* 1981). Neither Mirsalis *et al.* (1982) or Reitz *et al.* (1980) found any evidence that chloroform induced unscheduled DNA synthesis.

Two separate studies by Diaz Gomez and Castro (1980a, 1980b) were unable to detect binding of radiolabeled chloroform to DNA. DiRenzo *et al.* (1982) demonstrated, however, that metabolically activated chloroform can bind to DNA *in vitro* at very low levels. In the yeast *Saccharomyces cerevisiae*, exposure to chloroform was associated with an increase in the frequency of mitotic recombination and gene conversion (Callen *et al.*, 1980).

Kirkland *et al.* (1981) reported that exposure to chloroform induced a small increase in the frequency of sister chromatid exchange in cultured human lymphocytes. However, because no controls were used the significance of this report is questionable. Morimoto and Koizumi (1983) found that chloroform induced a concentration-dependent increase in sister chromatid exchange in cultured human lymphocytes and in bone-marrow cells from mice dosed by gavage.

There is little evidence that chloroform can induce sperm-head abnormalities in mice, but both reports are difficult to evaluate critically due to a lack of data presentation (Topham, 1980) or questionable statistical analysis (Land *et al.*, 1981).

Carcinogenicity in Animals

The earliest indication that chloroform might be carcinogenic to animals came in 1945 with publication of the results of a study by Eschenbrenner and Miller (1945). They administered a total of 30 doses of chloroform (128, 296, 592, 1,185, or 2,369 mg/kg) by gavage to strain A mice over 120 d. All animals in the highest dose group and all males from the next two highest dose groups died after receiving one or two doses of chloroform. Upon terminal sacrifice of the remaining animals, 4/4 and 3/3 females from the 1,185 and 592 mg/kg-groups, respectively, had hepatomas (Appendix Table D-9).

Rudali (1967) briefly described the results of a study that reportedly found evidence that chloroform was carcinogenic to NLC mice. Animals were given two doses a week of a 40% solution of chloroform for an unspecified period of time. At terminal sacrifice, only five of the original 24 animals survived; of these, three had hepatic tumors.

The NCI (1976b) conducted a bioassay in which chloroform was administered to B6C3F1 mice and Osborne-Mendel rats by gavage 5 d/wk for 78 wk. Mice received TWA doses of 138 or 277 mg/kg-d (males) and 238 or 477 mg/kg-d (females). Male rats were given doses of 90 or

180 mg/kg throughout the study period. Doses for female rats were initially set at 125 and 250 mg/kg, but were reduced after 22 wk, resulting in TWA doses of 100 and 200 mg/kg-d. In mice, treatment with chloroform induced a high incidence of hepatocellular carcinoma in animals of both sexes from all four treatment groups. The frequency of occurrence of these tumors was dose related and was significantly different from controls (Appendix Table D-9). Mortality among high-dose rats of both sexes was high. Only 28% survived until terminal sacrifice (compared with 44 to 48% survival in the low-dose groups). Male rats dosed with 180 mg/kg developed a significantly ($p = 0.016$) greater combined incidence of renal adenomas and adenocarcinomas than controls (Appendix Table D-10).

In 1979, Reuber published the results of his reexamination of the NCI (1976b) histology slides. Although he agreed with many of the findings of the NCI pathologists (i.e., that mice and rats had a high incidence of hepatic and renal tumors, respectively), some of his conclusions differed substantially. For example, Reuber determined that male mice from both treatment groups had a significantly higher incidence of malignant lymphoma (Appendix Table D-9) and that high- and low-dose male rats had significantly greater numbers of total malignant tumors than controls. Reuber also concluded that high-dose female rats had a significantly higher incidence of cholangiofibroma and cholangiocarcinoma relative to controls (Appendix Table D-10).

Roe *et al.* (1979) compared the carcinogenicity of chloroform administered in toothpaste to four separate strains of mice. (See Appendix Table D-9.) In the first of three separate experiments, two different doses of chloroform (17 or 60 mg/kg) were administered in toothpaste to ICI mice. By the 95th wk, eight high-dose male mice had developed renal tumors (five adenomas and three hypernephromas). The U.S. EPA (1985c) determined that the combined incidence of these tumors was significantly ($p < 0.001$) different from vehicle controls. A second experiment, also with ICI mice, utilized a single dose level of chloroform (60 mg/kg) administered in toothpaste with or without a variety of flavoring agents. As in the first experiment, a high incidence of renal adenomas and hypernephromas developed in chloroform-treated mice. The incidence of these tumors was significantly different from controls, whether evaluated alone or in combination. The final experiment, in which chloroform was administered in toothpaste to four separate strains of mice, failed to find any evidence of carcinogenicity. However, administration of 60 mg/kg-d of chloroform in arachis oil to male ICI mice resulted in a significant ($p < 0.01$) increase in the incidence of renal tumors.

Two separate publications of Tumasonis *et al.* (1985, 1987) described the results of a single study in which chloroform was administered to Wistar rats in drinking water. Over the 180-wk treatment period, males received TWA doses of 200 mg/kg-d and females, 240 mg/kg-d. At terminal sacrifice, Tumasonis *et al.* observed that ingestion of chloroform was associated with a statistically significant ($p < 0.03$) increase in the incidence of neoplastic nodules and hepatic adenofibrosis ($p < 0.001$) in females compared to untreated controls. A high incidence ($p < 0.001$) of hepatic adenofibrosis was also documented in treated males relative to controls.

Jorgenson *et al.* (1985) evaluated the carcinogenicity of chloroform administered in drinking water to female B6C3F1 mice and male Osborne-Mendel rats over 104 wk. Rats received TWA doses of 18, 38, 79, or 155 mg/kg-d; mice received TWA doses of 25, 50, 112, or 234 mg/kg-d (see DHS, 1988). In marked contrast to the results of the NCI (1976b) study, Jorgenson *et al.* found no evidence that chloroform was carcinogenic to female mice under the conditions of this

study. However, the incidence of renal tubular cell adenomas and the combined incidence of these adenomas and renal carcinomas were significantly ($p < 0.01$) greater in high dose rats than in controls. Total kidney tumors (adenomas, carcinomas, and nephroblastomas) were also significantly ($p < 0.01$) elevated in this group.

Although there appears to be ample evidence that chloroform is carcinogenic to animals, several investigations have failed to detect a carcinogenic response to treatment with chloroform. Palmer *et al.* (1979) administered chloroform by gavage (15, 75, or 165 mg/kg) to male and female Sprague-Dawley rats for approximately 1 y. Both control and treated animals developed severe respiratory and renal disease, resulting in the premature termination of the experiment. Histological examination of animals resulted in the identification of one malignant tumor in a chloroform-treated male rat and one benign tumor in a vehicle-control female. A subsequent study was also complicated by high mortality, and only 14 to 22% of controls and 26 to 32% of treated rats survived for the entire 95-wk study period. No significant differences in tumor incidence were noted between treated and control animals.

In an abstract, Roe *et al.* (1968) reported that subcutaneous injection of newborn C57xDBA2F1 mice with 200 mg of chloroform (one to eight times) did not appear to significantly affect the development of tumors. No evidence of carcinogenicity was found when beagle dogs were exposed to chloroform over a 7-y period (Appendix Table D-11) (Heywood *et al.*, 1979). Because the average lifespan of these animals is 13 to 14 y, the U.S. EPA (1985c) suggested that the treatment period may have been inadequate to evaluate the lifetime carcinogenic risk of chloroform to dogs.

The U.S. EPA (1985c) used tumor-incidence data from NCI (1976b), Roe *et al.* (1979), and Jorgenson *et al.* (1985) to calculate a series of alternative cancer potencies for metabolized chloroform. These values, and the associated tumor-incidence data, are listed in Appendix Table D-12. We note that our health risk assessment of chloroform was based on the median of these potency values, 0.028 $(mg/kg\text{-}d)^{-1}$ (see Table 5-13).

Carcinogenicity in Humans

Chloroform and other trihalomethanes are formed by the addition of chlorine to water that contains organic material. As a consequence, chloroform is ubiquitous in finished drinking water throughout the United States (Symons *et al.*, 1975). To address the potential health risk due to ingestion of chloroform-containing water, a number of epidemiological studies have examined the association between an increased risk of human cancer and exposure to chloroform (Wilkins *et al.*, 1979; Harris, 1974, Harris *et al.*, 1977; DeRouen and Diem, 1975; Page *et al.*, 1976; Salg, 1977; Cantor *et al.*, 1978; Hogan *et al.*, 1979; Young *et al.*, 1981; Brenniman *et al.*, 1978; Wilkins and Comstock, 1981; Lawrence *et al.*, 1984).

None of these studies have demonstrated a direct relationship between the presence of chloroform (or other trihalomethanes) in drinking water and an increased risk of cancer. Interpretation of data in these reports was frequently complicated by indirect and/or qualitative measurements, failure to control for smoking and alcohol consumption, and an inability to document actual exposures to chloroform. Although many studies were seriously compromised by these deficits, a number have shown a positive correlation between ingestion of chlorinated

drinking water and an increased risk of rectal, bladder, or colon cancer. However, the U.S. EPA (1985c) considered these data inadequate to evaluate the human carcinogenicity of chloroform.

1,1-dichloroethylene

Genotoxic Effects

The International Agency for Research on Cancer (IARC) concluded that there is "sufficient evidence" of 1,1-dichloroethylene's (1,1-DCE) activity in short-term tests of mutagenicity (IARC, 1982). This statement was based in part on observations by Bartsch *et al.* (1975) and Simmon *et al.* (1977) that 1,1-DCE was mutagenic to *Salmonella typhimurium.* Greim *et al.* (1975) also found positive evidence of 1,1-DCE's mutagenicity to *Escherichia coli* when tested in the presence of exogenous metabolic activation.

1,1-DCE induced point mutations and gene conversions in the yeast *Saccharomyces cerevisiae* (Bronzetti *et al.*, 1981), and bound to rat liver and kidney DNA *in vivo* and *in vitro* (Reitz *et al.*, 1980). 1,1-DCE was not mutagenic to V79 cells (Drevon and Kuroki, 1979) and did not induce dominant lethal mutations in rats or mice (Anderson *et al.*, 1977; Short *et al.*, 1977).

Carcinogenicity in Animals

Nine separate bioassays have been conducted to evaluate the carcinogenicity of 1,1-DCE to rats, mice, and hamsters. 1,1-DCE has been administered in drinking water, by gavage, inhalation, subcutaneous injection, or dermal application. These studies were critically evaluated by the U.S. EPA (1984, 1985d, 1985e). The reader is referred to these documents for a comprehensive review. In the course of these evaluations, the EPA determined that only one of the nine bioassays provided positive evidence of a carcinogenic effect (U.S. EPA, 1985e). The following discussion focuses only on the three studies which have been used by the U.S. EPA to calculate cancer potency (U.S. EPA, 1988b).

Quast *et al.* (1983) provided 1,1-DCE in drinking water *ad libitum* to male and female Sprague-Dawley rats for 2 y. The average concentrations of 1,1-DCE were 50, 100, or 200 ppm. The authors calculated that the TWA dose of 1,1-DCE ingested by males was 7, 10, or 20 mg/kg, and by females, 9, 14, or 30 mg/kg. Survival of animals was not substantially affected by 1,1-DCE, and approximately 50% of the animals remained alive after 20 months of treatment. Neither total tumor incidence, specific tumor incidence, nor the average number of tumors per animal was significantly altered by 1,1-DCE, except for mammary gland tumors in females. In the low-dose group, animals exposed to 1,1-DCE had a significantly ($p < 0.05$) higher incidence (40/48) of mammary gland fibroadenomas than in untreated controls (53/80). This finding was not considered to be biologically significant, however, because no dose-response relationship was observed, and the incidence of this tumor was within the range observed in historical controls.

The NTP reported the results of a bioassay in which 1,1-DCE was administered by gavage to F344 rats and B6C3F1 mice 5 d/wk for 104 wk (NTP, 1982). Rats were dosed with 1 or 5 mg/kg-d of 1,1-DCE, and mice received 2 or 10 mg/kg-d. An accident during the 82nd wk of

dosing killed 12 control and 10 low-dose male rats. Nevertheless, overall survival in rats was greater than 50% in all groups. In mice, survival of treated animals ranged from 64 to 84%. The initial statistical analysis of tumor-incidence data indicated that the incidence of adrenal pheochromocytomas, interstitial-cell tumors of the testes, islet-cell adenomas and carcinomas of the pancreas, and subcutaneous fibromas in male rats, pituitary adenomas in female rats, and lymphomas in female mice were significantly higher than in controls. Subsequent adjustment for intercurrent mortality showed that lymphomas (in low-dose female mice) were the only tumors whose incidence was actually significantly ($p < 0.05$) different from controls. However, a similar increase in lymphomas was not observed in high-dose female mice. The NTP (1982) concluded that 1,1-DCE was not carcinogenic to rats or mice under the conditions of this study. The U.S. EPA (1985e) noted that maximum tolerated doses may not have been used by the NTP, however, thereby limiting the ability of this study to detect a carcinogenic effect.

Maltoni *et al.* (1985, as cited in U.S. EPA, 1985e) exposed rats, mice, and hamsters to 1,1-DCE by inhalation 4 h/d, 5 d/wk for 12 months. Concentrations of 1,1-DCE were set at 10, 25, 50, 100, or 150 ppmv for rats, 10 or 25 ppmv for mice, and 25 ppmv for hamsters. A separate experiment examined the effects of 1,1-DCE administered by gavage to rats at 0.5, 5, 10, or 20 mg/kg-d, 4 or 5 d/wk for 52 wk. Terminal sacrifice took place between 121 and 157 wk depending on the species and route of administration.

Exposure of hamsters to 1,1-DCE had no significant effect on tumor incidence. The number of female rats with tumors (of any type) was significantly ($p < 0.05$) greater in animals exposed to 10 or 100 ppmv 1,1-DCE than in controls. Female rats from all exposure groups had significantly ($p < 0.05$) more mammary fibromas and fibroadenomas than controls, but the incidence of mammary carcinomas was higher in controls than in treated animals. Male and female mice exposed to 10 or 25 ppmv 1,1-DCE developed significantly ($p < 0.01$) more pulmonary adenomas than controls. Renal adenocarcinomas were found in significantly ($p < 0.01$) greater numbers in male mice exposed to 25 ppmv 1,1-DCE. The incidence of mammary carcinomas was significantly ($p < 0.01$) elevated in female mice exposed to 10 or 25 ppmv, relative to controls.

On the basis of the positive findings of the Maltoni *et al.* (1985) study, the U.S. EPA (1988b) classified 1,1-DCE as a Possible Human Carcinogen (Group C). The decision to place 1,1-DCE in Group C rather than in Group B (a Probable Human Carcinogen) was based in part on the weight of evidence of the negative findings from the eight other studies that have examined the carcinogenicity of 1,1-DCE (U.S. EPA, 1985e).

Nevertheless, the U.S. EPA (1988b) calculated cancer potencies for 1,1-DCE on the basis of tumor incidence data from Quast *et al.* (1983), NTP (1982), and Maltoni *et al.* (1985). This approach is unusual in that neither Quast *et al.* or the NTP found a statistically significant increase in the incidence of any tumor type.

Both the State of California (DHS, 1988) and the U.S. EPA (1988b) have derived drinking water standards for 1,1-DCE based on the results of a study (Quast *et al.*, 1983) that identified hepatic toxicity in female rats (midzonal fatty degeneration) as an endpoint, an approach which specifically ignores the data of Maltoni *et al.* (1985). The decision of the DHS to treat 1,1-DCE as a noncarcinogen was based on (1) the fact that the carcinogenicity data on 1,1-DCE are highly contradictory, and (2) State regulatory guidelines which stipulate that in order to regulate a substance as a potential human carcinogen on the basis of animal data, there must be "positive

evidence of carcinogenicity from properly conducted bioassays in two species of animals, or two properly conducted bioassays in the same species...." (DHS, 1985). Therefore, the positive findings of Maltoni *et al.* (1985) in mice, which have not been independently confirmed, do not constitute sufficient evidence of carcinogenicity. The U.S. EPA (1985e) apparently considered the data of Maltoni *et al.* (1985) inadequate to establish drinking water standards, and consequently, selected the results of a chronic toxicity in which 1,1-DCE was administered in drinking water.

Carcinogenicity in Humans

1,1-DCE is structurally related to a known human carcinogen, vinyl chloride, a fact which has contributed to concern over 1,1-DCE's potential carcinogenicity to humans. However, the only epidemiological study of human health risks from 1,1-DCE exposure is that of Ott *et al.* (1976). This study examined mortality among a group of 138 Dow Chemical Company workers exposed to 1,1-DCE for various periods of time. Ott *et al.* estimated TWA exposure concentrations of 1,1-DCE, and used these data in conjunction with the duration of exposure (in months) to calculate cumulative career exposures for workers. This calculation obscured crucial details and made it impossible to distinguish those workers who had been exposed to low concentrations of 1,1-DCE for a long period of time from those with higher exposures that may have occurred only for relatively brief intervals. Nonetheless, no significant differences in mortality were found between the exposed population and the controls. Although the study cohort was matched with controls with respect to age and smoking history, failure to account for exposure to other chemicals and the small size of the study group combined to make this study inadequate to evaluate the human carcinogenic potential of 1,1-DCE.

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Appendix Table D-1. Carcinogenicity bioassays for TCE administered by gavage to mice (from Bogen *et al.*, 1988).

Study	Strain	Sex	Dose	Tumor type	Incidence	Statistical significance[a]
NCI, 1976a	B6C3F1	M	0 mg/kg (matched)	Hepatocellular carcinoma	1/20	
			0 mg/kg (colony)		5/77	
			1,169 mg/kg[b]		26/50	p = 0.004
			2,339 mg/kg		31/48	p < 0.001
		F	0 mg/kg (matched)	Hepatocellular carcinoma	0/20	
			0 mg/kg (colony)		1/80	
			869 mg/kg[b]		4/50	p = 0.090
			1,739 mg/kg		11/47	p = 0.008
NTP, 1983	B6C3F1	M	0 mg/kg	Hepatocellular carcinoma	8/48	
			1,000 mg/kg[c]		30/50	p < 0.001
		M	0 mg/kg	Hepatocellular carcinoma or adenoma	11/48	
			1,000 mg/kg[c]		38/50	p < 0.001
		F	0 mg/kg	Hepatocellular carcinoma	2/48	
			1,000 mg/kg[c]		13/49	p < 0.05
		F	0 mg/kg	Hepatocellular adenoma	2/48	
			1,000 mg/kg[c]		8/49	p < 0.05
		F	0 mg/kg	Hepatocellular carcinoma or adenoma	4/48	
			1,000 mg/kg[c]		19/49	p < 0.001
Van Duuren *et al.*, 1979	ICR/Ha-Swiss	M	0 mg/kg			
			2.8 mg/kg[d]			[e]
		F	0 mg/kg			
			2.8 mg/kg[d]			[e]
Henschler *et al.*, 1984	ICR/Ha-Swiss	M	0 mg/kg			
			1,900 mg/kg[f]			[e]
			0 mg/kg			
		F	1,400 mg/kg[f]			[e]

[a]Fischer Exact Test using vehicle control group incidence unless otherwise noted.

[b]Five doses per week of industrial grade TCE [epichlorohydrin (0.09%); 1,2-epoxybutane (0.19%)] for 78 wk; observed for 12 wk; terminated on wk 90.

[c]Five doses per week of purified TCE for 103 wk; terminated between 103 and 107 wk.

[d]Dosed once a week for 89 wk with 0.5 g purified TCE.

[e]No statistically significant observed effect.

[f]Five doses per week of purified TCE for 78 wk; observed for 26 wk; terminated on wk 104.

Appendix Table D-2. Carcinogenicity bioassays for TCE administered by gavage to rats (from Bogen *et al.*, 1988).

Study	Strain	Sex	Dose	Tumor type	Incidence	Statistical significance[a]
NCI, 1976a	Osborne-Mendel	M	0 mg/kg			
			549 mg/kg[b]			([c])
		F	0 mg/kg			
			1,097 mg/kg[b]			([c])
NTP, 1983	F344/N	M	0 mg/kg	Renal tubular cell	0/48[d]	
			500 mg/kg[e]	adenocarcinoma	0/49	
			1,000 mg/kg		3/49	p = 0.028[f]
		M	0 mg/kg	Renal tubular cell adenoma/	0/48	
			500 mg/kg[e]	adenocarcinoma	2/49	
			1,000 mg/kg		3/49	p = 0.028[f]
		F	0 mg/kg			
			500 mg/kg[e]			([c])
			1,000 mg/kg			([c])
NTP, 1988	ACI 9935	M/F	0 mg/kg			
			500 mg/kg[g]			([c])
			1,000 mg/kg			([c])
	August 28807	M/F	0 mg/kg			
			500 mg/kg[g]			([c])
			1,000 mg/kg			([c])
	Marshall 520	M	0 mg/kg (untreated)	Testicular interstitial cell	16/46	
			0 mg/kg (vehicle)	tumor (almost exclusively	17/46	
			500 mg/kg[g]	benign)	21/48	
			1,000 mg/kg		32/48	p = 0.002
	Marshall 520	F	0 mg/kg			
			500 mg/kg[g]			([c])
			1,000 mg/kg			([c])
	Osborne-Mendel	M	0 mg/kg (untreated)	Renal tubular cell adenoma	0/50	
			0 mg/kg (vehicle)		0/50	
			500 mg/kg[g]		6/50	p = 0.007[h]
			1,000 mg/kg		1/50	
		M	0 mg/kg	Renal tubular cell adenoma/	0/50	
			500 mg/kg[g]	adenocarcinoma	6/50	p = 0.007[h]
			1,000 mg/kg		2/50	
		F	0 mg/kg			
			500 mg/kg[g]			([c])
			1,000 mg/kg			([c])

Appendix Table D-2. (Continued)

Study	Strain	Sex	Dose	Tumor type	Incidence	Statistical significance[a]
Maltoni *et al.*, 1986 BT 301	Sprague-Dawley	M	0 mg/kg			
			50 mg/kg[i]			(c)
			250 mg/kg			(c)
		F	0 mg/kg			
			50 mg/kg[i]			(c)
			250 mg/kg			(c)

[a]Fischer Exact Test unless otherwise noted.

[b]Five doses per week of industrial grade TCE [epichlorohydrin (0.09%); 1,2-epoxybutane (0.19%)] for 78 wk; observed for 32 wk; terminated on wk 110.

[c]No statistically significant observed effect.

[d]Terminal incidence (all tumors were observed upon terminal sacrifice at 103 wk).

[e]Five doses per week of purified TCE for 103 wk; terminated between 103 and 107 wk.

[f]Using "Life Table" or "Incidental Tumor" tests referenced in NTP (1983).

[g]Five doses per week of epoxide-free TCE for 103 wk; terminated between 110 and 111 wk.

[h]Using "Life Table" or "Incidental Tumor" tests referenced in NTP (1988).

[i]Four to five doses/wk of purified TCE for 52 wk (13 wk old at start).

Appendix Table D-3. Carcinogenicity bioassays for TCE based on inhalation in mice (from Bogen *et al.*, 1988).

Study	Strain	Sex	Dose	Tumor type	Incidence	Statistical significance[a]
Bell *et al.*, 1978[b]	B6C3F1	M	0 ppmv	Hepatocellular carcinoma	18/99	
			100 ppmv[c]		28/95	p = 0.046
			300 ppmv		31/100	p = 0.026
			600 ppmv		43/97	p < 0.001
		M	0 ppmv	Hepatocelluar adenoma	2/99	
			100 ppmv[c]		7/95	
			300 ppmv		7/100	
			600 ppmv		10/97	p = 0.015
		M	0 ppmv	Hepatocellular carcinoma or adenoma	20/99	
			100 ppmv[c]		35/95	p = 0.008
			300 ppmv		38/100	p = 0.004
			600 ppmv		53/97	p < 0.001

Appendix Table D-3. (Continued)

Study	Strain	Sex	Dose	Tumor type	Incidence	Statistical significance[a]
Bell *et al.*, 1978[b] (cont)		F	0 ppmv	Hepatocellular carcinoma or adenoma	8/99	
			100 ppmv[c]		9/100	
			300 ppmv		10/94	
			600 ppmv		17/99	p = 0.04
Henschler *et al.*, 1980	Han: NMRI	M	0 ppmv			
			100 ppmv[d]			(e)
			500 ppmv			(e)
		F	0 ppmv	Malignant lymphoma	9/29	
			100 ppmv[d]		17/30	p = 0.042
			500 ppmv		18/28	p = 0.012
Fukuda *et al.*, 1983	ICR	F	0 ppmv	Lung adenocarcinoma	1/49	
			50 ppmv[f]		3/50	
			150 ppmv		8/50	p < 0.05
			450 ppmv		7/46	p < 0.05
Maltoni *et al.*, 1986 BT 303	Swiss	M	0 ppm			
			100 ppm[g]			(e)
			600 ppm			(e)
		F	0 ppm			
			100 ppm[g]			(e)
			600 ppm			(e)
Maltoni *et al.*, 1986 BT 305	Swiss	M	0 ppm	Pulmonary tumors	10/90	
			100 ppm[h]	(almost exclusively benign)	11/90	
			300 ppm		23/90	p < 0.05
			600 ppm		27/90	p < 0.01
		M	0 ppm	Hepatomas (malignant)	4/90	
			100 ppm[h]		2/90	
			300 ppm		8/90	
			600 ppm		13/90	p < 0.05
		F	0 ppm			
			100 ppm[h]			(e)
			300 ppm			(e)
			600 ppm			(e)

Appendix Table D-3. (Continued)

Study	Strain	Sex	Dose	Tumor type	Incidence	Statistical significance[a]
Maltoni *et al.*, 1986	B6C3F1	F	0 ppm	Pulmonary tumors	4/90	
			100 ppm[i]	(almost exclusively benign)	6/90	
	BT 306/306 bis		300 ppm		7/90	
			600 ppm		15/90	$p < 0.05$
		M	0 ppm			
			100 ppm[i]			(e)
			300 ppm			(e)
			600 ppm			(e)
		M and F,	0 ppm	Hepatomas (malignant)	4/180	
		combined	100 ppm[i]		5/180	
			300 ppm		7/180	
			600 ppm		15/180	$p < 0.01$

[a]Fischer Exact Test against control incidence, unless otherwise noted.

[b]From U.S. EPA, 1985a, pp. 8-42.

[c]Six h/d, 5 d/wk, technical grade TCE [epichlorohydrin (0.09%)] for 104 wk.

[d]Six h/d, 5 d/wk, purified TCE for 78 wk; observed for 52 wk; terminated on wk 130 (median survival time of controls = approx. 104 wk).

[e]No statistically significant observed effect.

[f]Seven h/d, 5 d/wk, reagent grade TCE [carbon tetrachloride (0.128%); benzene (0.019%); epichlorohydrin (0.019%); 1,1,2-trichloroethane (0.010%)] for 104 wk; terminated on wk 107.

[g]Seven h/d, 5 d/wk, epoxide-free TCE for 8 wk (11 wk old at start).

[h]Seven h/d, 5 d/wk, epoxide-free TCE for 78 wk (11 wk old at start), observed to wk 145 (most animals dead by wk 104).

[i]Seven h/d, 5 d/wk, epoxide-free TCE for 78 wk (12 wk old at start), observed to wk 145 (most males and females dead by wk 90 and 130, respectively).

Appendix Table D-4. Carcinogenicity bioassays for TCE based on inhalation in rats and hamsters (from Bogen *et al.*, 1988).

Study	Strain	Sex	Concentration	Tumor type	Incidence	Statistical significance[a]
Bell *et al.*, 1978	Charles River	M/F	0 ppmv			
			100 ppmv[b]			(c)
			300 ppmv			(c)
			600 ppmv			(c)
Henschler *et al.*, 1980	Han:WIST	M/F	0 ppmv			
			100 ppmv[d]			(c)
			500 ppmv			(c)
Henschler *et al.*, 1980	Syrian hamster	M/F	0 ppmv			
			100 ppmv[e]			(c)
			500 ppmv			(c)
Fukuda *et al.*, 1983	Sprague-Dawley	F	0 ppmv			
			50 ppmv[f]			(c)
			150 ppmv			(c)
			450 ppmv			(c)
Maltoni *et al.*, 1986 BT 302	Sprague-Dawley	M	0 ppm			
			100 ppm[g]			(c)
			600 ppm			(c)
		F	0 ppm			
			100 ppm[g]			(c)
			600 ppm			(c)
Maltoni *et al.*, 1986	Sprague-Dawley	M	0 ppmv	Testes Leydig-cell tumor (almost exclusively benign)	6/135 BT 304/304 bis	
			100 ppm[h]		16/130	$p < 0.05$
			300 ppm		30/130	$p < 0.01$
			600 ppm		31/130	$p < 0.01$
		F	0 ppmv			
			100 ppm[h]			(c)
			300 ppm			(c)
			600 ppm			(c)

Appendix Table D-4. (Continued)

[a]Fischer Exact Test unless otherwise noted.

[b]Six h/d, 5 d/wk, technical grade TCE [epichlorohydrin (0.09%)] for 104 wk.

[c]No statistically significant observed effect.

[d]Six h/d, 5 d/wk, purified TCE for 78 wk; observed for 78 wk; terminated on wk 156.

[e]Six h/d, 5 d/wk, purified TCE for 78 wk; observed for 52 wk; terminated on wk 130.

[f]Seven h/d, 5 d/wk, reagent grade TCE [carbon tetrachloride (0.128%); benzene (0.019%); epichlorohydrin (0.019%); 1,1,2-trichloroethane (0.010%)] for 104 wk; terminated on wk 107.

[g]Seven h/d, 5 d/wk, epoxide-free TCE for 8 wk.

[h]Seven h/d, 5 d/wk, epoxide-free TCE for 104 wk.

Appendix Table D-5. Carcinogenicity bioassays for TCE administered to mice dermally, subcutaneously, and in drinking water (from Bogen *et al.*, 1988).

Study	Strain	Sex	Dose or concentration	Tumor type	Incidence	Statistical significance[a]
Van Duuren *et al.*, 1979	Ha ICR	M/F	0 mg			
			1.0 mg[b]			(c)
Van Duuren *et al.*, 1979	Ha ICR	M/F	0 mg			
			0.5 mg[d]			(c)
Herren-Freund *et al.*, 1987	B6C3F1	M/F	0 mg/L			
			3 mg/L[e,f]			(c)
			40 mg/L[g]			(c)

[a]Fischer Exact Test unless otherwise noted.

[b]Three dermal applications/wk of purified TCE for 83 wk.

[c]No statistically significant observed effect.

[d]One subcutaneous injection/wk of purified TCE for 89 wk.

[e]Exposed via drinking water for 61 wk; terminated on wk 65.

[f]Equivalent to 0.59 mg/kg-d, assuming a daily water intake of 6.3 mL/d for mice weighing 32 g.

[g]Equivalent to 7.7 mg/kg-d, assuming a daily water intake of 6.7 mL/d for mice weighing 32 g.

Appendix Table D-6. Carcinogenicity bioassays for the TCE metabolites, TCA and DCA, administered to mice in drinking water (from Bogen *et al.*, 1988).

Study	Strain	Sex	Dose or concentration	Tumor type	Incidence	Statistical significance[a]
Herren-Freund *et al.*, 1987	B6C3F1	M	0 mg DCA/L	Hepatocellular carcinoma	0/22	
			5,000 mg DCA/L[b,c]		21/26	$p < 0.01$
		M	0 mg DCA/L	Hepatocellular adenoma	2/22	
			5,000 mg DCA/L[b,c]		25/26	$p < 0.01$
Herren-Freund *et al.*, 1987	B6C3F1	M	0 mg TCA/L	Hepatocellular carcinoma	0/22	
			5,000 mg TCA/L[b,c]		7/22	$p < 0.01$
		M	0 mg TCA/L	Hepatocellular adenoma	2/22	
			5,000 mg TCA/L[b,c]		8/22	$p < 0.01$

[a]Fischer Exact Test unless otherwise noted.

[b]TCA = trichloracetic acid, DCA = dichloroacetic acid. Exposed via drinking water for 61 wk; terminated on wk 65.

[c]Equivalent to 1,000 mg/kg-d, assuming a daily water intake of 6.0 mL/d for 30-g mice.

Appendix Table D-7. Dose-response data and corresponding estimates of carcinogenic potency for metabolized TCE (from Bogen *et al.*, 1988).

Study species strain	Sex	Tumor		95% UCL potency[c] $(mg/kg\text{-}d)^{-1}$	
		Type[a]	Incidence[b]	BW[d]	SA[e]
NCI (1976a) Mice B6C3F1	M	HCC	1/20		
			26/48	0.0025	0.032
			31/40		
			0/18		
	F	HCC	4/42	0.00073	0.0098
			11/37		
NTP (1983) Mice B6C3F1	M	HCC	8/48	0.0019	0.023
			30/50		
		HCC or HCA	11/48	0.0029	0.036
			38/50		
	F	HCC	2/41		
			13/41	0.00096	0.012

Appendix Table D-7. (Continued)

Study species strain	Sex	Tumor Type[a]	Tumor Incidence[b]	95% UCL potency[c] $(mg/kg\text{-}d)^{-1}$ BW[d]	95% UCL potency[c] $(mg/kg\text{-}d)^{-1}$ SA[e]
NTP (1983) Mice B6C3F1 (Continued)					
		HCC or HCA	4/41	0.0014	0.018
			19/41		
NTP (1983) Rats F344/N	M		0/33		
		RTC	0/20	0.00074	0.0043
			3/16		
			0/45		
		RTC or RTA	2/39	0.00065	0.0038
			3/26		
Bell *et al.* (1978) Mice B6C3F1	M		18/99		
			28/95		
		HCC	31/100	0.0020	0.026
			43/97		
			20/99		
		HCC or HCA	35/95	0.0028	0.036
			38/100		
			53/97		
Fukuda *et al.* (1983) Mice ICR	F		1/49		
			3/50		
		LA	8/50	0.0014	0.019
			7/46		
Maltoni *et al.* (1986) Mice Swiss	F		4/90		
			2/90		
		MH	8/90	0.00082	0.0098
			13/90		

[a]HCC = hepatocellular carcinoma, HCA = hepatocellular adenoma, RTC = renal tubular-cell adenocarcinoma, RTA = renal tubular-cell adenoma, ML = malignant lymphoma, LA = lung adenocarcinoma, MH = malignant hepatoma.

[b]Tumor-incidence denominator excludes animals dying before the occurrence of the first corresponding tumor type observed in the NCI (1976a) and NTP (1983) studies.

[c]"Potency" here means the low-dose dose-response slope expressed by an upper-bound linear multistage coefficient such that at very low doses, risk = (potency × metabolized dose), according to a multistage risk prediction model (U.S. EPA, 1980; Anderson *et al.*, 1983; Crump and Howe, 1984). 95% UCL = one-tailed 95% upper confidence limit.

[d]BW = Body weight interspecies dose-extrapolation method; equivalent doses assumed to be in mg/kg.

[e]SA = Surface area interspecies dose-extrapolation method; equivalent doses assumed to be in $mg/kg^{2/3}$.

Appendix Table D-8. Summary of bioassay tumor-incidence data and corresponding estimates of cancer potency of metabolized PCE (from Bogen *et al.*, 1987).

Study	Species (strain)	Sex	Concentration or dose	Tumor		95% UCL potency[c] $(mg/kg\text{-}d)^{-1}$	
				Type[a]	Incidence[b]	BW[d]	SA[e]
NCI (1977)	Mice (B6C3F1)	M	0 mg/kg-d		2/17		
			536 mg/kg-d	HC	32/49	0.032	0.42
			1072 mg/kg-d		27/48		
		F	0 mg/kg-d		2/20		
			386 mg/kg-d	HC	19/48	0.022	0.31
			772 mg/kg-d		19/48		
NTP (1986)	Mice (B6C3F1)	M	0 ppmv		7/49		
			100 ppmv	HC	25/49	0.015	0.19
			200 ppmv		26/50		
			0 ppmv		16/49		
			100 ppmv	HAC	8/49	0.024	0.30
			200 ppmv		18/50		
		F	0 ppmv		1/48		
			100 ppmv	HC	13/50	0.0073	0.095
			200 ppmv		36/50		
			0 ppmv		3/48		
			100 ppmv	HAC	6/50	0.0098	0.13
			200 ppmv		2/50		
NTP (1986)	Rats (F344/N)	M	0 ppmv		28/50		
			200 ppmv	MLK	37/50	0.064	0.35
			400 ppmv		37/50		
		F	0 ppmv		18/50		
			200 ppmv	MLK	30/50	0.040	0.24
			400 ppmv		29/50		

[a]HC = hepatocellular carcinoma; HAC = hepatocellular adenoma; and MLK = mononuclear cell leukemia.

[b]"Potency" here means the low-dose dose-response slope expressed by an upper-bound linear multistage coefficient such that at very low doses risk = (potency × metabolized dose), according to a multistage risk prediction model (U.S. EPA, 1980; Anderson *et al.*, 1983; Crump and Howe, 1984). 95% UCL = one-tailed 95% upper confidence limit.

[c]BW = Body weight interspecies dose-extrapolation method; equivalent doses assumed to be in mg/kg.

[d]SA = Surface area interspecies dose-extrapolation method; equivalent doses assumed to be in $mg/kg^{2/3}$.

Appendix Table D-9. Carcinogenicity bioassays of chloroform administered orally to mice.

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
Eschenbrenner and Miller (1945)	A	M	0	Hepatoma	0/5	
			148[b]		0/5	
			296		0/3	
			592		(c)	
			1,185		(c)	
			2,369		(c)	
		F	0	Hepatoma	0/5	
			148		0/5	
			296		0/5	
			592		3/3	$p = 0.018$[d]
			1,185		4/4	$p = 0.0080$[d]
			2,369		(c)	
NCI (1976b)	B6C3F1	M	0 (colony)	Hepatocellular carcinoma	5/77	
			0 (match)		1/18	
			138[e]		18/47	$p = 0.011$[f]
			277		44/45	$p = 3.1 \times 10^{-10}$
		F	0 (colony)	Hepatocellular carcinoma	1/80	
			0 (match)		0/19	
			238		36/45	$p = 4.0 \times 10^{-10}$
			477		39/41	$p = 3.7 \times 10^{-14}$
Reuber (1979)	B6C3F1	M	0	Hyperplastic hepatic nodule	1/17	
			0 (match)		2/17	
			138[g]		11/46	$p = 0.25$
			277		0/44	n.d.
		M	0	Small hepatocellular carcinoma	1/17	
			0 (match)		0/17	
			138		3/46	$p = 0.38$
			277		3/44	$p = 0.37$
		M	0	Large hepatocellular carcinoma	1/17	
			0 (match)		0/17	
			138		17/46	$p = 0.00018$
			277		41/44	$p < 0.00001$

Appendix Table D-9. (Continued)

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
Reuber (1979) (Continued)		M	0	Hepatocellular carcinoma or hyperplastic nodule	3/17	
			0 (match)		2/17	
			138		31/46	$p < 0.00001$
			277		44/44	$p < 0.00001$
		M	0	Malignant lymphoma	0/17	
			0 (match)		0/17	
			138		14/46	$p = 0.0064$
			277		10/44	$p = 0.028$
		F	0	Hyperplastic hepatic nodule	0/20	
			0 (match)		0/19	
			238		1/45	n.d.
			477		0/40	n.d.
		F	0	Small hepatocellular carcinoma	1/17	
			0 (match)		0/17	
			238		3/45	$p = 0.34$
			477		1/40	$p = 0.68$
		F	0	Large hepatocellular carcinoma	0/20	
			0 (match)		0/19	
			238		37/45	$p < 0.00001$
			477		39/40	$p < 0.00001$
		F	0	Hepatocellular carcinoma or hyperplastic nodule	0/20	
			0 (match)		0/19	
			238		41/45	$p < 0.00001$
			477		40/40	$p < 0.00001$
		F	0	Malignant lymphoma	0/20	
			0 (match)		0/19	
			238		9/45	$p = 0.032$
			477		4/40	$p = 0.20$

Appendix Table D-9. (Continued)

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
Roe *et al.* (1979)	ICI	M	0	Renal adenoma	0/72	
			17[h]		0/37	
			60		5/38	p < 0.05[i]
		M	0	Renal hypernephroma	0/72	
			17		0/37	
			60		3/38	p < 0.01[i]
		M	0	Renal adenoma or hypernephroma	0/72	
			17		0/37	
			60		8/38	p < 0.001[i]
		F	0			
			17			
			60			(j)
		M	0	Renal adenoma	1/45	
			0 (match)		6/237	
			60[k]		7/49	p < 0.05[i]
		M	0	Renal hypernephroma	0/45	
			0 (match)		0/237	
			60		2/49	p < 0.01[i]
		M	0	Renal adenoma or hypernephroma	1/45	
			0 (match)		6/237	
			60		9/49	p < 0.001[i]
		M	0	Renal adenoma or hypernephroma	0/83	
			0 (match)		1/49	
			60[l]		5/47	p = 0.093
		M	0	Renal adenoma or hypernephroma	1/50	
			60[m]		12/48	p < 0.001[i]
	C57BL	M	0			
			60[k]			(j)
	CBA	M	0			
			60[k]			(j)
	CF/1	M	0			
			60[k]			(j)

Appendix Table D-9. (Continued)

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
Jorgenson *et al.* (1985)	B6C3F1	F	0			
			0 (match)			
			25[n]			
			50			
			112			
			234			(j)

[a]Fischer Exact Test unless otherwise noted.

[b]Thirty doses of chloroform in olive oil, administered by gavage over 120 d. Terminal sacrifice at 150 d.

[c]All animals in treatment group died within first 8 d of treatment.

[d]No statistical analysis of data provided. Values based on our calculations.

[e]Five doses/wk of USP grade chloroform (>99.0% purity) by gavage, in corn oil for 78 wk. Terminal sacrifice at 92 wk.

[f]Values from U.S. EPA (1985c).

[g]Data cited under Reuber (1979) are based on a reevaluation of the NCI (1976b) data. Dosing protocol as listed in ([e]).

[h]Chloroform administered by gavage in a toothpaste base, 6 d/wk for 80 wk. Terminal sacrifice at wk 96.

[i]Values from U.S. EPA (1985c); statistical test not specified.

[j]No statistically significant effect.

[k]Chloroform administered as in ([h]). Terminal sacrifice at wk 104.

[l]Chloroform administered as in ([h]). Terminal sacrifice at wk 97 or 99.

[m]Chloroform administered in arachis oil by gavage, 6 d/wk for 80 wk. Terminal sacrifice at wk 97 or 99.

[n]Pesticide-quality chloroform purified by distillation twice weekly, supplied *ad libitum* in drinking water for 104 wk. Terminal sacrifice at wk 104. Matched control animals were restricted to the water intake of the highest dose group. TWA doses are from DHS (1988).

Appendix Table D-10. Carcinogenicity bioassays of chloroform administered orally to rats.

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
NCI (1976b)	Osborne-Mendel	M	0 (colony)	Renal carcinoma or adenoma	0/99	
			0 (match)		0/19	
			90[b]		4/38	$p = 0.27$[c]
			180		12/27	$p = 0.014$
		F	0 (colony)	Thyroid adenoma or carcinoma	1/98	
			0 (match)		1/18	
			100		8/35	$p < 0.05$
			200		10/35	$p < 0.05$

Appendix Table D-10. Carcinogenicity bioassays of chloroform administered orally to rats.

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
Reuber (1979)[d]	Osborne-Mendel	M	0 (colony)	Hyperplastic hepatic nodule	0/20	
			0 (match)		1/19	
			90[b]		5/50	p = 0.47
			180		8/49	p = 0.22
		M	0 (colony)	Hepatocellular carcinoma	0/20	
			0 (match)		0/19	
			90		0/50	
			180		2/49	p = 0.081
		M	0 (colony)	Hepatocellular carcinoma or hyperplastic nodule	0/99	
			0 (match)		1/19	
			90[b]		5/50	p = 0.44
			180		10/49	p = 0.045
		M	0 (colony)	All liver tumors	0/99	
			0 (match)		2/19	
			90		5/50	p = 0.71
			180		12/49	p = 0.049
		M	0 (colony)	Renal adenoma or carcinoma	0/99	
			0 (match)		0/19	
			90		8/50	p = 0.06
			180		14/49	p = 0.0049
		M	0 (colony)	Total malignant tumors	6/99	
			0 (match)		5/19	
			90		25/50	p = 0.031
			180		21/49	p = 0.16
		F	0 (colony)	Hepatocellular carcinoma or hyperplastic nodule	1/20	
			0 (match)		2/20	
			100		9/39	p = 0.20
			200		14/39	p = 0.031
		F	0 (colony)	Cholangio-fibroma or cholangio-carcinoma	0/20	
			0 (match)		0/20	
			100		3/39	p = 0.28
			200		11/39	p = 0.0060
		F	0 (colony)	All liver tumors	1/20	
			0 (match)		2/20	

Appendix Table D-10. Carcinogenicity bioassays of chloroform administered orally to rats.

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
			100		10/39	p = 0.14
			200		20/39	p = 0.002
		F	0 (colony)	Thyroid adenoma or carcinoma	3/20	
			0 (match)		1/20	
			100		11/39	p = 0.037
			200		12/39	p = 0.021
		F	0 (colony)	Hyperplastic hepatic nodule	1/20	
			0 (match)		2/20	
			100		7/39	p = 0.35
			200		12/39	p = 0.036
		F	0 (colony)	Hepatocellular carcinoma	0/20	
			0 (match)		0/20	
			100		2/39	p = 0.43
			200		2/39	p = 0.43
Palmer *et al.* (1979)	Sprague-Dawley	M	0			
			15[e]			
			75			
			165			(f)
		F	0			
			15			
			75			
			165			(f)
		M	0			
			60[g]			(f)
		F	0			
			60			(f)
Tumasonis *et al.* (1985)	Wistar	M	0[h]	Hepatic adenofibrosis[i]	0/22	
			200		17/28	p < 0.001
		F	0	Hepatic adenofibrosis	0/18	
			240		34/40	p < 0.001
		F	0	Neoplastic nodules	0/18	
			240		10/40	p < 0.03
Jorgenson *et al.* (1985)	Osborne-Mendel	M	0[j]	Lymphomas and leukemias	5/303	
			0 (match)		1/50	
			18		19/316	p < 0.01
			38		5/148	n.d.
			79		2/48	n.d.

Appendix Table D-10. Carcinogenicity bioassays of chloroform administered orally to rats.

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance[a]
Jorgenson *et al.* (1985) (cont)			155		3/50	$p < 0.01$[k]
		M	0	All circulatory system tumors	5/303	
			0 (match)		0/50	
			18		6/316	n.d.
			38		3/148	n.d.
			79		3/48	$p < 0.01$[k]
			155		3/50	$p < 0.05$[k]
		M	0	Renal adenoma	4/301	
			0 (match)		0/50	
			18		2/313	n.d.
			38		3/148	n.d.
			79		2/48	n.d.
			155		5/50	$p < 0.01$[k]
		M	0	Renal adenoma or carcinoma	4/301	
			0 (match)		1/50	
			18		4/313	n.d.
			38		4/148	n.d.
			79		3/48	n.d.
			155		7/50	$p < 0.01$[k]
		M	0	All renal tumors	5/301	
			0 (match)		1/50	
			18		6/313	n.d.
			38		7/148	n.d.
			79		3/48	n.d.
			155		7/50	$p < 0.01$[k]

[a]Fischer Exact Test unless otherwise noted.

[b]Five doses/wk of USP grade chloroform (>99.0% purity) by gavage in corn oil, for 78 wk. Terminal sacrifice after 111 wk.

[c]Values from U.S. EPA (1985c).

[d]Data cited under Reuber (1979) are from a re-evaluation of the NCI (1976b) data. Dosing protocol as listed in (b).

[e]Six doses/wk of chloroform administered in a toothpaste base, by gavage. Study terminated after 52 wk.

[f]No statistically significant effect.

[g]Dosing protocol as in ([d]) except that treatment continued for 80 wk. Terminal sacrifice at wk 95.

[h]Chloroform supplied to animals in drinking water, *ad libitum*, for 180 wk. Timing of terminal sacrifice not specified, but assumed to have been at wk 180. Quantities of chloroform are time-weighted average daily doses calculated from data presented by Tumasonis *et al.* (1985; 1987).

[i]Hepatic adenofibrosis is a proliferative lesion of the bile duct, which has also been classified as cholangiocellular carcinoma (Schauer and Kunze, 1979).

[j]Pesticide-quality chloroform purified by distillation twice weekly, supplied *ad libitum* in drinking water for 104 wk. Terminal sacrifice at wk 104. Matched control animals were restricted to the water intake of the highest dose group. TWA daily doses listed are based on a recalculation of Jorgenson *et al.* (1985). See DHS (1988).

[k]Peto trend test.

Appendix Table D-11. Carcinogenicity bioassays of chloroform administered orally to dogs.

Study	Strain	Sex	Experimental applied dose (mg/kg-d)	Tumor type	Incidence	Statistical significance
Heywood *et al.* (1979)	Beagle	M	0			
			15[a]			
			30			(b)
		F	0			
			15			
			30			(b)

[a]Six doses/wk of chloroform for 374 wk. Administered in a toothpaste base, in gelatin capsules.

[b]No statistically significant effect.

Appendix Table D-12. Estimates of the carcinogenic potency of metabolized chloroform.[a]

Study species strain	Sex	Tumor Type[b]	Tumor Incidence[c]	95% UCL potency[d] $(mg/kg\text{-}d)^{-1}$
NCI (1976b) Mice B6C3F1	M		1/18	
		HCC	18/50	0.033
			44/45	
	F		0/20	
		HCC	36/45	0.20
			39/41	
NCI (1976b) Rats Osborne-Mendel	M	RA or RC	0/19	
			4/50	0.024
			12/50	
Roe *et al.* (1979) Mice ICI	M	RA or RH	0/50	
			9/48	0.10
Jorgenson *et al.* (1985) Rats Osborne-Mendel	M		4/301	
		RA or RC	4/313	
			4/148	0.0044
			3/48	
			7/50	
	M	RA,	1/50	
		RC, and NB	6/313	
			7/148	0.0061
			3/48	
			7/50	

Appendix Table D-12. (Continued)

[a]Information derived from U.S. EPA (1985c), Appendix Tables 8-20, 8-21, 8-22, 8-23, and 8-24.

[b]HCC = hepatocellular carcinoma, RA = renal adenoma, RC = renal carcinoma, RH = renal hypernephroma, NB = nephroblastoma.

[c]Tumor-incidence denominator appears not to have been adjusted by EPA for animals dying before the occurrence of the first corresponding tumor type observed in the NCI (1976b) study.

[d]"Potency" here means the low dose, dose-response slope expressed by an upper-bound linear multistage coefficient such that at very low doses, risk = potency × metabolized dose, according to a multistage risk prediction model (U.S. EPA, 1980; Anderson *et al.*, 1983). 95% UCL = one-tailed 95% upper confidence limit. The U.S. EPA's intended unit for these reported potency values is ambiguous, but these potency values are clearly equivalent to ones based on a surface-area method for interspecies extrapolation of equipotent doses under the assumption that humans would metabolize 100% of very small ingested doses.

Appendix E

Risk and Hazard Estimates Based on EPA Methodology for Ground Water Contaminants

Appendix E

Risk and Hazard Estimates Based on EPA Methodology for Ground Water Contaminants

D. W. Layton

The exposure and risk assessments presented in Section 5 differ in several respects from EPA methods, as described in U.S. EPA (1986). For example, we used cancer potencies for TCE, PCE, and chloroform that were based on metabolized, rather than applied doses. In addition, our review of the toxicological data base on 1,1-DCE indicates that it should be treated as a noncarcinogen (see Appendix D). The EPA, in contrast, has classified 1,1-DCE as a Class C carcinogen and has published cancer potencies for it (U.S. EPA, 1988). We calculated water-based exposures to the VOCs of concern for four different pathways: water ingestion, inhalation of VOCs volatilized into shower air, dermal uptake of VOCs in bath water, and the ingestion of garden fruits and vegetables irrigated with contaminated well water. The EPA normally uses two pathways: water ingestion and inhalation of VOCs in shower air. The EPA also assumes that exposure from the inhalation pathway is equivalent to the ingestion pathway.

To compare results of the two approaches, we calculated cancer risks using EPA methodology and toxicity data. Appendix Tables E-1 and E-2 present the predicted cancer risks for the best-estimate and health-conservative exposure scenarios. Both tables include the total risks calculated using LLNL methodology. Finally, we compared the predicted exposures to safe intake levels (defined by RfD values in the IRIS data base; U.S. EPA, 1988) by computing the ratio of RfD to predicted exposure. These results are presented in Appendix Tables E-3 and E-4 for the two exposure scenarios.

By EPA methodology, the highest cancer risk for the best estimate case is 3×10^{-6}, associated primarily with 1,1-DCE exposure at well BF. The LLNL maximum risk for that case was 2×10^{-7} associated with chloroform at well AF.

Appendix E References

U.S. Environmental Protection Agency (U.S. EPA) (1986), *Superfund Public Health Evaluation Manual*, U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C. (EPA/540/1-86/060, OSWER Directive 9285.4-1).

U.S. Environmental Protection Agency (U.S. EPA) (1988), *Environmental Protection Agency's Integrated Risk Information System (IRIS)—An Electronic Data Base Containing Health Risk and U.S. EPA Regulatory Information on Specific Chemicals*, U.S. Environmental Protection Agency, Office of Research and Development, Environmental Criteria and Assessment Office, Cincinnati, Ohio (March 1988).

Appendix Table E-1. Predicted cancer risks for the best-estimate exposure scenario based on EPA methodologies. Total risks for the oral and inhalation pathways are compared with the risks presented in Table 5-14.

Well/VOC	Concentration (mg/L)	Oral intake[a] (mg/kg-d)	Oral potency[b] $(mg/kg-d)^{-1}$	Cancer risk	Inhalation potency[c] $(mg/kg-d)^{-1}$	Cancer risk	EPA total risk[d]	LLNL total risk[e]
Well A-F								
Chloroform	1.5E–04	4.3E–06	6.1E–03	3E–08	8.1E–02	3E–07	4E–07	2E–07
Well B-F (Time period of max TCE)								
TCE	1.0E–04	2.9E–06	1.1E–02	3E–08	1.3E–02	4E–08	7E–08	9E–08
Other VOCs	1.0E–05							
1,1-DCE	5.6E–06	1.6E–07	6.0E–01	1E–07	1.2E+00	2E–07	3E–07	
Carbon tetrachloride	4.4E–06	1.3E–07	1.3E–01	2E–08	1.3E–01	2E–08	4E–08	4E–08
						Sums =	4E–07	1E–07
Well B-F (Time period of max other VOCs)								
TCE	8.0E–05	2.3E–06	1.1E–02	2E–08		3E–08	5E–08	7E–08
Chloroform	1.0E–05	2.9E–07	6.1E–03	2E–09	8.1E–02	2E–08	2E–08	1E–08
Other VOCs	2.0E–05							
1,1-DCE	1.1E–05	3.1E–07	6.0E–01	2E–07	1.2E+00	4E–07	6E–07	
Carbon tetrachloride	9.0E–06	2.6E–07	1.3E–01	3E–08	1.3E–01	3E–08	6E–08	8E–08
						Sums =	7E–07	2E–07
Well C-F								
PCE	1.0E–05	2.9E–07	5.1E–02	1E–08	3.3E–03	9E–10	2E–08	7E–08

[a]Oral intakes are calculated for a reference 70-kg person consuming 2 L of water per day.

[b]The oral cancer potency is from U.S. EPA (1988).

[c]The inhalation cancer potency is from U.S. EPA (1988). The cancer risk for inhaling VOCs in shower air is calculated by multiplying the oral intake, which is assumed equal to inhalation exposure, by the inhalation potency factor.

[d]Total risk is the sum of the cancer risks for the oral and inhalation pathways.

[e]From Table 5-14.

Appendix Table E-2. Predicted cancer risks for the health-conservative exposure scenario based on EPA methodologies. Total risks for the oral and inhalation pathways are compared with the risks presented in Table 5-15.

Well/VOC	Concentration (mg/L)	Oral intake[a] (mg/kg-d)	Oral potency[b] $(mg/kg-d)^{-1}$	Cancer risk	Inhalation potency[c] $(mg/kg-d)^{-1}$	Cancer risk	EPA total risk[d]	LLNL total risk[e]
Well A-N								
PCE	6.2E-03	1.8E-04	0.051	9E-06	0.0033	6E-07	1E-05	4E-05
TCE	2.6E-01	7.4E-03	0.011	8E-05	0.013	1E-04	2E-04	2E-04
Chloroform	5.5E-02	1.6E-03	0.0061	1E-05	0.081	1E-04	1E-04	8E-05
Other VOCs	1.8E-02							
1,1-DCE	1.0E-02	2.9E-04	0.6	2E-04	1.2	3E-04	5E-04	
Carbon tetrachloride	7.9E-03	2.3E-04	0.13	3E-05	0.13	3E-05	6E-05	7E-05
						Sums =	9E-04	4E-04
Well B-N								
PCE	5.2E-02	1.5E-03	0.051	8E-05	0.0033	5E-06	8E-05	4E-04
TCE	4.7E-01	1.3E-02	0.011	1E-04	0.013	2E-04	3E-04	4E-04
Chloroform	2.0E-02	5.7E-04	0.0061	3E-06	0.081	5E-05	5E-05	3E-05
Other VOCs	4.2E-02	1.2E-03						
1,1-DCE	2.4E-02	6.7E-04	0.6	4E-04	1.2	8E-04	1E-03	
Carbon tetrachloride	1.8E-02	5.3E-04	0.13	7E-05	0.13	7E-05	1E-04	2E-04
						Sums =	2E-03	1E-03
Well C-N								
PCE	2.7E-01	7.7E-03	0.051	4E-04	0.0033	3E-05	4E-04	2E-03
TCE	6.2E-02	1.8E-03	0.011	2E-05	0.013	2E-05	4E-05	6E-05
Chloroform	8.2E-03	2.3E-04	0.0061	1E-06	0.081	2E-05	2E-05	1E-05
Other VOCs	2.5E-02							
1,1-DCE	1.4E-02	4.0E-04	0.6	2E-04	1.2	5E-04	7E-04	
Carbon tetrachloride	1.1E-02	3.1E-04	0.13	4E-05	0.13	4E-05	8E-05	8E-05
						Sums =	1E-03	2E-03

Appendix Table E-2. (Continued)

Well/VOC	Concentration (mg/L)	Oral intake[a] (mg/kg-d)	Oral potency[b] $(mg/kg-d)^{-1}$	Cancer risk	Inhalation potency[c] $(mg/kg-d)^{-1}$	Cancer risk	EPA total risk[d]	LLNL total risk[e]
Well A-M								
PCE	5.9E–03	1.7E–04	0.051	9E–06	0.0033	6E–07	9E–06	4E–05
TCE	2.4E–01	6.9E–03	0.011	8E–05	0.013	9E–05	2E–04	2E–04
Chloroform	4.9E–02	1.4E–03	0.0061	9E–06	0.081	1E–04	1E–04	7E–05
Other VOCs	1.8E–02							
1,1-DCE	1.0E–02	2.9E–04	0.6	2E–04	1.2	3E–04	5E–04	
Carbon tetrachloride	7.9E–03	2.3E–04	0.13	3E–05	0.13	3E–05	6E–05	7E–05
						Sums =	9E–04	4E–04
Well B-M								
PCE	5.2E–02	1.5E–03	0.051	8E–05	0.0033	5E–06	8E–05	4E–04
TCE	3.8E–01	1.1E–02	0.011	1E–04	0.013	1E–04	3E–04	4E–04
Chloroform	1.7E–02	4.9E–04	0.0061	3E–06	0.081	4E–05	4E–05	3E–05
Other VOCs	4.0E–02							
1,1-DCE	2.2E–02	6.4E–04	0.6	4E–04	1.2	8E–04	1E–03	
Carbon tetrachloride	1.8E–02	5.0E–04	0.13	7E–05	0.13	7E–05	1E–04	2E–04
						Sums =	2E–03	1E–03
Well C-M								
PCE	2.1E–01	6.0E–03	0.051	3E–04	0.0033	2E–05	3E–04	1E–03
TCE	5.8E–02	1.7E–03	0.011	2E–05	0.013	2E–05	4E–05	5E–05
Chloroform	7.3E–03	2.1E–04	0.0061	1E–06	0.081	2E–05	2E–05	1E–05
Other VOCs	2.6E–02							
1,1-DCE	1.5E–02	4.2E–04	0.6	2E–04	1.2	5E–04	7E–04	
Carbon tetrachloride	1.1E–03	3.3E–04	0.13	4E–05	0.13	4E–05	9E–05	1E–04
						Sums =	1E–03	2E–03
Well A-F								
PCE	6.1E–03	1.7E–04	0.051	9E–06	0.0033	6E–07	9E–06	4E–05
TCE	2.3E–01	6.6E–03	0.011	7E–05	0.013	9E–05	2E–04	2E–04
Chloroform	4.5E–02	1.3E–03	0.0061	8E–06	0.081	1E–04	1E–04	7E–05
Other VOCs	1.8E–02							
1,1-DCE	1.0E–02	2.9E–04	0.6	2E–04	1.2	3E–04	5E–04	
Carbon tetrachloride	7.9E–03	2.3E–04	0.13	3E–05	0.13	3E–05	6E–05	7E–05
						Sums =	9E–04	4E–04

Appendix Table E-2. (Continued)

Well/VOC	Concentration (mg/L)	Oral intake[a] (mg/kg-d)	Oral potency[b] $(mg/kg-d)^{-1}$	Cancer risk	Inhalation potency[c] $(mg/kg-d)^{-1}$	Cancer risk	EPA total risk[d]	LLNL total risk[e]
Well B-F								
PCE	4.9E–02	1.4E–03	0.051	7E–05	0.0033	5E–06	8E–05	3E–04
TCE	3.4E–01	9.7E–03	0.011	1E–04	0.013	1E–04	2E–04	3E–04
Chloroform	1.6E–02	4.6E–04	0.0061	3E–06	0.081	4E–05	4E–05	2E–05
Other VOCs	3.5E–02							
1,1-DCE	2.0E–02	5.6E–04	0.6	3E–04	1.2	7E–04	1E–03	
Carbon tetrachloride	1.5E–02	4.4E–04	0.13	6E–05	0.13	6E–05	1E–04	1E–04
						Sums =	1E–03	7E–04
Well C-F								
PCE	1.7E–01	4.9E–03	0.051	2E–04	0.0033	2E–05	3E–04	1E–03
TCE	5.6E–02	1.6E–03	0.011	2E–05	0.013	2E–05	4E–05	5E–05
Chloroform	6.9E–03	2.0E–04	0.0061	1E–06	0.081	2E–05	2E–05	1E–05
Other VOCs	2.4E–02							
1,1-DCE	1.3E–02	3.8E–04	0.6	2E–04	1.2	5E–04	7E–04	
Carbon tetrachloride	1.1E–03	3.0E–04	0.13	4E–05	0.13	4E–05	8E–05	1E–04
						Sums =	1E–03	1E–03

[a]Oral intakes are calculated for a reference 70-kg person consuming 2 L of water per day.

[b]The oral cancer potency is from U.S. EPA (1988).

[c]The inhalation cancer potency is from U.S. EPA (1988). The cancer risk for inhaling VOCs in shower air is calculated by multiplying the oral intake, which is assumed equal to inhalation exposure, by the inhalation potency factor.

[d]Total risk is the sum of the cancer risks for the oral and inhalation pathways.

[e]From Table 5-15.

Appendix Table E-3. Analysis of the noncarcinogenic risks of the principal VOCs for the best-estimate exposure scenario.

Well/VOC	Concentration[a] (mg/L)	Water ingestion[b] (mg/kg-d)	Oral RfD[c] (mg/kg-d)	Ratio[d]
Well A-F				
Chloroform	1.5E–04	4.3E–06	1E–02	4.3E–04
Well B-F				
TCE	1.0E–04	2.9E–06		
Other VOCs				
1,1-DCE	5.6E–06	1.6E–07	9E–03	1.8E–05
Carbon tetrachloride	4.4E–06	1.3E–07	7E–04	1.8E–04
			Sum =	2.0E–04
Well B-F				
Chloroform	2.0E–05	5.7E–07	1E–02	5.7E–05
Other VOCs				
1,1-DCE	4.5E–05	1.3E–06	9E–03	1.4E–04
Carbon tetrachloride	3.5E–05	1.0E–07	7E–04	1.4E–03
			Sum =	1.6E–03
Well C-F				
PCE	1.0E–05	2.9E–07	1E–02	2.9E–05

[a]Concentrations are from Table 4-3.

[b]Ingestion exposures are calculated for a reference 70-kg person consuming 2 L of water per day.

[c]The oral reference doses (RfD) are from U.S. EPA (1988). The oral RfD for TCE is not available at this time.

[d]The hazard index is computed by taking the sum of the ratios of the calculated exposure divided by the RfD value.

Appendix Table E-4. Analysis of the noncarcinogenic risks of the principal VOCs for the health-conservative exposure scenario.

Well/VOC	Concentration[a] (mg/L)	Water ingestion[b] (mg/kg-d)	Oral RfD[c] (mg/kg-d)	Ratio[d]
Well A-N				
PCE	6.2E-03	1.8E-04	1.0E-02	2E-02
TCE	2.6E-01	7.4E-03		
Chloroform	5.5E-02	1.6E-03	1.0E-02	2E-01
Other VOCs	1.8E-02			
1,1-DCE	1.0E-02	2.9E-04	9.0E-03	3E-02
Carbon tetrachloride	7.9E-03	2.3E-04	7.0E-04	3E-01
			Sum =	5E-01
Well B-N				
PCE	5.2E-02	1.5E-03	1.0E-02	1E-01
TCE	4.7E-01	1.3E-02		
Chloroform	2.0E-02	5.7E-04	1.0E-02	6E-02
Other VOCs	4.2E-02	1.2E-03		
1,1-DCE	2.4E-02	6.7E-04	9.0E-03	7E-02
Carbon tetrachloride	1.8E-02	5.3E-04	7.0E-04	8E-01
			Sum =	1E+00
Well C-N				
PCE	2.7E-01	7.7E-03	1.0E-02	8E-01
TCE	6.2E-02	1.8E-03		
Chloroform	8.2E-03	2.3E-04	1.0E-02	2E-02
Other VOCs	2.5E-02			
1,1-DCE	1.4E-02	4.0E-04	9.0E-03	4E-02
Carbon tetrachloride	1.1E-02	3.1E-04	7.0E-04	4E-01
			Sum =	1E+00
Well A-M				
PCE	5.9E-03	1.7E-04	1.0E-02	2E-02
TCE	2.4E-01	6.9E-03		
Chloroform	4.9E-02	1.4E-03	1.0E-02	1E-01
Other VOCs	1.8E-02			
1,1-DCE	1.0E-02	2.9E-04	9.0E-03	3E-02
Carbon tetrachloride	7.9E-03	2.3E-04	7.0E-04	3E-01
			Sum =	5E-01

Appendix Table E-4. (Continued)

Well/VOC	Concentration[a] (mg/L)	Water ingestion[b] (mg/kg-d)	Oral RfD[c] (mg/kg-d)	Ratio[d]
Well B-M				
PCE	5.2E–02	1.5E–03	1.0E–02	1E–01
TCE	3.8E–01	1.1E–02		
Chloroform	1.7E–02	4.9E–04	1.0E–02	5E–02
Other VOCs	4.0E–02			
1,1-DCE	2.2E–02	6.4E–04	9.0E–03	7E–02
Carbon tetrachloride	1.8E–02	5.0E–04	7.0E–04	7E–01
			Sum =	1E+00
Well C-M				
PCE	2.1E–01	6.0E–03	1.0E–02	6E–01
TCE	5.8E–02	1.7E–03		
Chloroform	7.3E–03	2.1E–04	1.0E–02	2E–02
Other VOCs	2.6E–02			
1,1-DCE	1.5E–02	4.2E–04	9.0E–03	5E–02
Carbon tetrachloride	1.1E–02	3.3E–04	7.0E–04	5E–01
			Sum =	1E+00
Well A-F				
PCE	6.1E–03	1.7E–04	1.0E–02	2E–02
TCE	2.3E–01	6.6E–03		
Chloroform	4.5E–02	1.3E–03	1.0E–02	1E–01
Other VOCs	1.8E–02			
1,1-DCE	1.0E–02	3.2E–04	9.0E–03	3E–02
Carbon tetrachloride	7.9E–03	1.9E–04	7.0E–04	3E–01
			Sum =	5E–01
Well B-F				
PCE	4.9E–02	1.4E–03	1.0E–02	1E–01
TCE	3.4E–01	9.7E–03		
Chloroform	1.6E–02	4.6E–04	1.0E–02	5E–02
Other VOCs	3.5E–02			
1,1-DCE	2.0E–02	5.6E–04	9.0E–03	6E–02
Carbon tetrachloride	1.5E–02	4.4E–04	7.0E–04	6E–01
			Sum =	9E–01

Appendix Table E-4. (Continued)

Well/VOC	Concentration[a] (mg/L)	Water ingestion[b] (mg/kg-d)	Oral RfD[c] (mg/kg-d)	Ratio[d]
Well C-F				
PCE	1.7E–01	4.9E–03	1.0E–02	5E–01
TCE	5.6E–02	1.6E–03		
Chloroform	6.9E–03	2.0E–04	1.0E–02	2E–02
Other VOCs	2.4E–02			
1,1-DCE	1.3E–02	3.8E–04	9.0E–03	4E–02
Carbon tetrachloride	1.1E–02	3.0E–04	7.0E–04	4E–01
			Sum =	1E+00

[a]Concentrations are from Table 4-4.

[b]Ingestion exposures are calculated for a reference 70-kg person consuming 2 L of water per day.

[c]The oral reference doses (RfD) are from U.S. EPA (1988). The oral RfD for TCE is not available at this time.

[d]The hazard index is computed by taking the sum of the ratios of the calculated exposure divided by the RfD value.

LAWRENCE LIVERMORE
NATIONAL LABORATORY
University of California • Livermore, California • 94551